THE
# VETERINARY JOURNAL

**Editor :**
FREDERICK HOBDAY, C.M.G., F.R.C.V.S., F.R.S.E.,
HONORARY VETERINARY SURGEON TO HIS MAJESTY THE KING,
OFFICIER DU MÉRITE AGRICOLE (FRANCE),
CAVALIERE DEI SS. MAURIZIO E LAZZARO (ITALY),
HONORARY MEMBER OF THE AMERICAN VETERINARY ASSOCIATION,
PRINCIPAL OF THE ROYAL VETERINARY COLLEGE, LONDON.

**Sub-Editor :**
GLADSTONE MAYALL, M.R.C.V.S.

MAY, 1932.

COLLEGE OF AGRICULTURE AND LIBRARY JUN 4

## VETERINARY RESEARCH.

THE continued response to the Editorial request for articles on sheep diseases has been such that this present issue of THE VETERINARY JOURNAL is a worthy supplement to the special April quarterly number, and will be a further stimulus to the efforts of the Veterinary Committee of the Agricultural Research Council in their promised efforts to endeavour to provide the veterinary profession with its much belated funds whereby its researchers' brains may have the opportunity to put their well thought out themes into practical shape—an opportunity which has only been held back by miserable parsimony and lack of funds. It has been an astonishing error of judgment in the past, and it is still an amazing fact, that those in authority over attempts to control animal disease in this country seem so willing to spend such enormous sums of the taxpayers' money in *compensation* for animals killed as actually suffering from certain diseases, and so little in efforts to discover and *prevent* their causation.

The young graduate who is willing (and capable) to follow research into the causation and prevention of animal diseases receives such miserable financial and other compensations as compared with what he can make in general practice and other spheres of veterinary outlook, that he has no alternative but to set a research career on one side ; and it is only in rare instances that he returns to it, which is not only a loss to the profession, but to the country at large. *The labourer is worthy of his hire ; the remedy lies in a fair deal.* As we have many times repeated before, if those who are vitally concerned—*i.e.*, the owners of stock and the Government—will provide a fair and just financial outlook, the profession will find not only the men, but the brains.

## RINDERPEST IN INDIA.

IT has been estimated that about a quarter of a million cattle die annually of rinderpest in India. This estimated loss is, perhaps, small when it is realised that there are nearly two hundred million cattle, including buffaloes, in the



XUM

sub-continent, and that a large proportion of these cattle are of very low value, allowed to live largely through the religious prejudices of the people against destruction of useless animals, and exposed, in a country where provision is not made against seasonal fodder shortage, to death from starvation. Moreover, the indigenous cattle of the plains of India possess a degree of resistance against fatal rinderpest which is high in comparison with that of our Western breeds of cattle. The real loss from the ever-imminent menace of the disease seems to be, nevertheless, a much larger one than the proportionate annual loss from actual mortality would imply. It has undoubtedly discouraged attempts at improvement of the indigenous cattle by selective breeding, produced much loss among cattle debilitated through attack, even when the outcome of infection has not been death, and caused much misery from its periodic visitations among a people whose occupations are mainly rural and who are dependent almost entirely upon cattle for the pursuit of their relatively primitive agricultural operations. These facts have all been recently well presented in the Report of the Royal Commission on Agriculture in India (1928).

The Civil Veterinary Departments of the several provinces in India are mainly engaged in combating rinderpest. Hitherto, the control of the disease has been attempted almost entirely by inoculating cattle exposed to infection in the scene of outbreaks with anti-rinderpest serum. This serum has been made and sold in increasing quantities for several years for use throughout India from the veterinary laboratory situated at Muktesar in the Himalayan foothills. The protection conferred by the serum lasts only a few days, but it is hoped that when the cattle are under this protection they will contract infection naturally by contact from diseased cattle in the neighbourhood, and thus obtain the durable immunity, lasting many years, which results from recovery from the disease. This system of inoculation by means of serum alone is, however, not beyond serious reproach, especially when it is realised that in the conditions of field work in India effective contact at the opportune moment with virulent infection may not be established, or the necessary measures of isolation and control of movement in an infected area, which would render the serum protection by itself definitely effective, cannot be undertaken. We have heard it recently declared by expert authority that for these reasons the large volume of serum now used at great cost to inoculate cattle in India is largely wasted.

A much more satisfactory protection is conferred by the serum-simultaneous method of inoculation. In this method the cattle are inoculated with serum, nd at the same time with the living virus of rinderpest contained in the blood taken from cattle at the height of the disease. This method has been practised in several countries from which rinderpest is otherwise difficult to eradicate, and has succeeded very well, particularly in Egypt. Unfortunately, it is not devoid of danger, but on the whole the accidental mortality may be said to amount to rather less that one per cent. of the cattle inoculated. This method has not been practised as a wholesale measure of protection on a considerable scale until very recently in India. A step forward in making the method safer and more

XUM

easy of application in that country was reported by Dr. Edwards (1928), from the Muktesar laboratory, when he showed that the rinderpest virus could be adapted in a special way by passage to goats, and that in these animals the virus underwent a marked attenuation in virulence for cattle. This decrease in virulence was such that cattle inoculated with it passed through a mild attack of rinderpest and recovered, although they did not receive a simultaneous injection of serum. He (Dr. Edwards), however, did not advise at that time the abandonment of the simultaneous serum protection entirely in the conditions of field practice.

In our present number, an interesting article is published by Major R. F. Stirling, Director of Veterinary Services, Central Provinces, India, in which he reports the successful immunisation of over five hundred cattle by inoculation with the goat-adapted virus alone. His experience had shown that the serum-simultaneous method of inoculation was prohibitive in his province on account of the high cost of the serum prescribed, and with commendable enterprise he and his staff proceeded to investigate cautiously the possibility of using the goat virus alone. The merits of this method, if it should prove on more extended trial to be applicable generally with safety to indigenous cattle in India, would seem to lie in the ease with which it can be applied to confer a *durable* immunity, and the cost of the materials required, which is relatively negligible. There is nothing novel in the principle of the method, as it is analogous to the now well-established method of immunising human beings against small-pox with the cow-pox virus; and to the recently introduced method, which is said to be giving highly satisfactory results, of immunising fowls against fowl-pox with the pigeon-pox virus. The attempts made in recent years to obtain a satisfactory " dead " vaccine for immunisation on a large scale in field conditions have been, it is feared, disappointing; the statement made by the present director (Mr. F. Ware) of the Muktesar Laboratory (Annual Report for year ending March, 1931), that so far the experiments carried out at that laboratory have not proved that the vaccines used in other countries are as safe or efficient as the serum-simultaneous method, as practised in India, may be taken as a trustworthy indication of the value of these vaccines. There is also some evidence in the same report to substantiate the conclusions now reached by Major Stirling, that the living goat virus may be safely inoculated alone for the immunisation of Indian cattle.

The Royal Commission on Agriculture in India were probably right in concluding that rinderpest and other contagious diseases in India must in the main be combated by measures which aim at protecting the individual animal rather than by measures which aim at stamping out the source of infection. Seeing that the position regarding widespread individual protection in Indian conditions now seems to be that serum inoculation alone is costly and of very limited efficacy; the serum-simultaneous method of inoculation is prohibitive in cost, but efficacious; and the recent attempts at preparing a satisfactory " dead " vaccine have been disappointing; the main hope of success would now seem to lie in inoculation with a cheap, readily available, living, attenuated virus, along the lines reported by Major Stirling in his preliminary announcement.

XUM

# General Articles

## THE ÆTIOLOGY OF BRAXY-LIKE DISEASES OF SHEEP.

By A. D. McEWEN, B.Sc., M.R.C.V.S.
*South-Eastern Agricultural College, Wye, Kent.*

THIS short article is the expression of a personal conception of the status of our knowledge regarding the ætiology of certain acute diseases of sheep which are attributed to the pathogenic anaerobic bacteria, where the bacteria or their toxins are supposed to have entered the body by way of the alimentary canal. Diseases of lambs are not considered, nor yet the diseases of sheep caused through the contamination of external wounds.

Where the Scottish name braxy, or the North European terms bradsot or braasot are in use, either of these terms would be the popular name given to the type of disease which it is proposed to discuss. Geographical incidence has, however, prevented the constant use of these terms. Thus we find that in Australia a disease of sheep is popularly known as black disease, although the veterinarians who first described this disease, and were conversant with the literature of the time on braxy, called it a braxy-like disease, and considered it likely to be the same as braxy. Again, the term " struck " is applied to certain acute diseases of sheep in the Romney Marsh district. There is every likelihood that had a Scottish, Icelandic, or Norwegian sheep farmer observed black disease in Australia, or " struck " on the Romney Marsh, he would have named it braxy, bradsot, or braasot, as the case might be. For convenience of expression, the term braxy-like disease will be used in this article, but when this is done, it is only to convey by three words the idea of an acute and fatal disease of sheep which may be caused by anaerobic bacteria.

Two essentials should be fulfilled before an investigation on the ætiology of any of these braxy-like diseases can yield reliable information. The most important of these is the examination of carcases immediately after death, before post-mortem proliferation of bacteria or putrefactive changes occur. The second is a bacteriological technique which will enable a worker to give to an incriminated micro-organism a description of sufficient accuracy so that it may be identified and recognised by others. These two essentials were almost totally disregarded by earlier workers. That descriptions of anaerobic bacteria were inadequate was due to the lack of technique prevailing at the time, but the general use of putrefying carcases for investigational purposes was less excusable. In some of the earlier papers definite conclusions were based upon scanty studies on a few specimens from putrefying carcases. The value of such work is often negligible, therefore a number of papers commonly referred to in articles on these

XUM

diseases will not be considered here because their information is worthless from a scientific standpoint. Quite apart from such papers, the choice of references is selective and not exhaustive, and the more recent works referred to have been taken because of their direct bearing upon the particular subject at issue.

The difficulty in obtaining suitable material for investigation is often very real, and this more than any other factor retards progress. The braxy-like diseases of sheep are more or less seasonal in incidence, and often peculiar to certain localities, and at first sight it would appear that no difficulty should be experienced in finding animals before or at the time of death. But this is not so. Affected animals seldom exhibit noticeable symptoms until within a few hours of death, and the investigator must be prepared to seek for his material, and to consider himself fortunate when an animal is found suffering from the disease, or at the time of death, and the carcase can be post-mortemed whilst perfectly fresh. With the braxy-like diseases, symptoms, when seen, merely indicate the grave condition of the animal, and that death is approaching. The symptoms are of no differential diagnostic value in distinguishing one disease from another.

Black disease of sheep in Australia, to give it the popular name, is the disease upon which information is most complete. Yet the first scientifically valuable report of this disease is of such recent origin as 1918. Prior to 1918, it may be taken that more was known concerning braxy or bradsot, and it appeared probable that black disease would be shown to be the same disease as braxy. Braxy or bradsot was generally regarded as an entity, though in Scotland the possibility of different forms of braxy had been suggested, and workers in Germany had queried the similarity of the bradsot of that country with the bradsot of Iceland and Norway. The latest work indicates that in Europe two separate braxy-like diseases exist, and that one of these resembles black disease. Researches into the ætiology of these diseases being historically intermingled, it will be convenient to follow these in more or less chronological order.

The first investigations to give a scientific conception of the ætiology of bradsot were undertaken by Nielsen.[17] Neilsen worked in Iceland and in Norway, and published his findings in 1880, and amplified these in 1897. He regarded the characteristic lesions of the disease as consisting of hæmorrhagic areas of the mucosa of the abomasum, caused by an invasion of these parts by an anaerobic bacterium which occurred singly, in pairs, or even in chain formation, and which formed spores whilst in the tissues. The bacilli were not confined to the local lesions, but were found in other organs of the body, and in the blood stream, often in great numbers. Death was attributed to a toxæmia, and generalised infection with the bacilli. Nielsen did not examine animals slaughtered whilst suffering from the disease, nor carcases immediately after death. Nevertheless, he considered that the bacillus which he found was the cause of bradsot, and that the large numbers found in the body a short time after death excluded the possibility of their being mere post-mortem invaders.

XUM

In commenting on the occurrence of the disease, he notes the great number of cases which appear in October, and suggests that this is due to three factors: Firstly, the greater concentration of the animals, owing to these being brought in from the hill pasture to the immediate neighbourhood of the farmhouse, thus exposing them to a more massive infection, particularly on account of the careless and unhygienic manner in which bradsot carcases were disposed of. Secondly, as sheep were but recently shorn, they would be subject to the debilitating influence of cold. Finally, the ingestion of frozen food so chilled the walls of the abomasum that the organ became a prey to the bacteria.

Neilsen's experimental data are most interesting. Tissues of sheep which had died of bradsot when inoculated into mice and guinea-pigs, generally, but not invariably, killed these animals; but Nielsen experienced great difficulty in producing a fatal disease in the sheep by the subcutaneous inoculation of such material. As a rule a circumscribed œdema and infiltration of the inoculated part which dispersed was all that occurred. It was not until 1895 that he succeeded in obtaining virulent material capable of killing a sheep in one to two days when inoculated subcutaneously, and the lesions were essentially those of a gas gangrene infection of the inoculated leg. When mice and guinea-pigs succumbed to inoculation they did not show a blood stream infection, and it was only from the inoculation site that bacilli were recoverable. Neilsen's description of the cultural characters of the micro-organisms he worked with are of little value. They were, however, anaerobic, and further, there was a marked loss of virulence shown by the bacilli after they had been cultivated.

About the time that Nielsen published his second article, Jensen[7] (1896) recorded his investigations on the disease. He made no field investigations, but confined himself to laboratory work, cultivating anaerobic bacilli from material sent from Iceland, which in some cases was very putrid, and in others it had been dried on glass slides. He also cultivated similar bacilli from a sheep's stomach which had been pickled in alcohol. He compared the bacilli he isolated from these most questionable sources with bacilli isolated by Nielsen, and considered these to be similar and the cause of bradsot.

The bacillus now called the bradsot bacillus he compared with the Rauschbrand (black quarter) bacillus and a so-called malignant œdema bacillus, and in deep agar the bradsot bacillus was found to be the most delicately growing micro-organism of the three. The question may be asked: what organisms did Jensen call the Rauschbrand and the malignant œdema bacilli? The identity of the later would be difficult to place; but there is a reasonable supposition that the Rauschbrand bacillus was *B. chauvœi*, as presumably it had been isolated from a bovine suffering from black quarter. But he recorded the fact that the former grew more abundantly than the bradshot bacillus in deep agar. Now *B. chauvœi* is a notoriously timid grower in deep agar. Therefore either Jensen's Rauschbrand bacillus was not *B. chauvœi*, or the bradsot bacillus was an exceedingly exacting micro-organism in that medium. Jensen, in an inadequately

XUM

controlled experiment, reported that sheep immune to bradsot were susceptible to Rauschbrand, and thus the bacteria may be differentiated. He attempted to produce the disease by feeding sheep with the bacteria, and by feeding thistles and bacteria : his results were negative, but the amounts fed, and the number of sheep used, are not given.

These papers by Nielsen and by Jensen gave a preliminary interest to the problem of bradsot. Thereafter a number of workers isolated bacteria from carcases of sheep. These they called the bradsot bacillus ; and some unsuccessfully attempted to reproduce the disease by feeding the bacilli to sheep. On account of the type of material investigated, the work is of little value. But a report which appeared in 1906 will be quoted, not because of any intrinsic merit, though it was as thorough and extensive an investigation of braxy as any of its time, but on account of certain interesting paragraphs. The work is the Report of the Departmental Committee on Louping Ill and Braxy, 1906,[18] and the paragraph, page 151, is as follows : " The disease is known in the West Highlands as ' braxy,' and in the broad acceptation of the term covers several diseases of the sheep which break out in the late autumn and early winter months, and which have certain features in common. There are at least four such diseases, probably even more, two of which have apparently never been identified ; they have their equivalents in what are known by shepherds and others as ' the red braxy,' 'the white braxy,' 'the water braxy,' etc. Each is caused by a specific anaerobe, which most likely in the whole of them gains entrance to the body of the sheep through the stomach and intestine." Does truth lie behind this paragraph, or is it mere nonsense ?

Two more quotations from the Report have a bearing upon the question under consideration. Page 180 : " There is a certain waywardness and uncertainty in the inoculation of braxy, as there is in the case of all the members of this group of diseases. Thus, in the early stages of Professor Hamilton's study of the malady, before this committee was appointed, he brought home to Aberdeen, time and time, from the West Highlands, blood, peritoneal liquid and viscera from braxy animals, and endeavoured to inoculate these by various experiments on Aberdeenshire sheep, but without success." Again, under the heading— " Inoculability of Animals other than Sheep "—we have the following sentence, page 193 : " Our own experience, however, of comparative inoculability is that, as in the case of the sheep itself, there is a peculiar uncertainty in the result."

In 1908 and 1909, Miessner[12, 13], as a result of his own observations on fresh cadavers, ventured to doubt the conclusions of previous workers on the grounds that researches made on the so-called bradsot bacilli were obtained from carcases where putrefactive changes had commenced, and that the bacillus was in all probability a post-mortem invader. These bacilli had never been shown to produce the disease, even when amounts of one litre were fed to sheep, and the inoculation disease did not resemble the natural form of bradsot. Further, if animals said to be affected with bradsot

XUM

were slaughtered and examined, or examined at the time of natural death, reddened and swollen areas of the abomasum and congested parts of the duodenum were found, but not as constant features. On the other hand, the peritoneal sac was invariably distended with fluid, and frequently the liver showed dark-red areas with yellowish centres varying in size up to a few centimetres in diameter. In the fresh cadavers investigated by Miessner, no bacteria were present in the blood or organs except in the liver lesions, but from decomposed carcases bacilli indistinguishable from the bradsot bacillus were recovered. Miessner therefore concluded that these so-called bradsot bacilli were saprophytic cadaver bacilli, and doubted their ætiological significance. Miessner's observations were of great service, and by challenging the methods of his predecessors he was the first to demand the more exacting procedure in investigations which were necessary before research could progress with assurance.

Jensen[8] (1915) felt bound to combat the heterodox views of Miessner, particularly as they cast doubt upon the propriety of the vaccination methods which he had devised. After several years, during which time he had collected material from Icelandic sheep slaughtered whilst suffering from bradsot, he was able to publish the histo-pathological appearances of the abomasal lesions presented by ten cases. They showed a remarkably constant picture, consisting of necrosis and hæmorrhagic inflammation of the mucosa, together with an inflammatory œdema of the sub-mucosal tissues. The diseased mucosa and sub-mucosa were invaded by the bradsot bacilli, the bacilli being particularly plentiful in the latter site, where they were often found in felted masses. Infiltrating from the healthy to the diseased tissue were numerous leucocytes. The bacilli were also demonstrated microscopically in the kidney and liver in a number of cases. Jensen concluded that the lesions in the abomasum presented the features of a primary infection, and that the bacilli seen in the lesions were ante-mortem invaders, and the cause of the disease. He agrees that the bradsot bacilli may be found as post-mortem invaders of the body, but contended that this did not prejudice the case for their incrimination as the cause of the disease. Discussing the disease described by Miessner, he does not consider this the same as the Icelandic form, although he believes the latter exists in Mecklenberg. These 1915 observations of Jensen are of considerable significance: they give the first clear picture of the histo-pathology of lesions from fresh cases of a braxy-like disease of sheep. The bacilli were not identified by any cultural work, but the article loses little or nothing by this omission, descriptions of bacilli having hitherto generally added to the confusion of the muddled mixing of the anaerobic bacteria.

The most thorough and critical investigations yet written regarding the ætiology of braxy-like diseases of sheep now appeared. Dodd[2, 3] (1918 and 1921) published a summary of his researches on black disease in New South Wales. These two papers cover continuous investigations from 1914 to 1921, and are a record of the most painstaking, thorough, and critical researches. In contra-distinction to the work of the majority of those who had written upon the braxy-

XUM

like diseases of sheep, Dodd's work may appear to have suffered from too rigorous a refusal to give a ready acceptance to evidence of bacterial infection, while any possibility of post-mortem or even ante-mortem secondary bacterial invasion remained. But this highly critical attitude enabled him to place the theory of the ætiology of black disease upon a foundation which subsequent workers have slightly amplified, rearranged in some minor matters, but otherwise confirmed. In his earliest investigations he worked with material from animals found dead, and from such material he obtained pathogenic anaerobic bacteria. But recognising the imperative necessity of investigating fresh cases, he at times camped out for several days together in order to follow the flocks, and obtain living cases. As a result, he was able, in 1918, to report upon the detailed examinations of twenty sheep which had been examined at the time of death. Material from these cases was fed to sheep, and was inoculated into sheep and laboratory animals, and examined histologically. The feeding experiments were negative, the inoculation experiments were generally negative, in the majority of cases no bacteria being recovered from the tissues of affected sheep, and those bacteria occasionally encountered were not constant in pathogenicity nor yet in species. Histological examinations yielded most interesting findings, but before mentioning these, the macroscopic appearance of the black disease lesions should be noted. The most characteristic lesions were found in the liver, and consisted of one or more necrotic foci up to ½ in. to 2 in. in diameter, usually circular in outline and sharply defined. These had a yellowish centre, and were surrounded by a reddened zone. Hæmorrhagic areas in the liver were sometimes found. The pericardial sac was distended with pale-coloured fluid. The abomasum and duodenum frequently showed areas of congestion, the latter sometimes hæmorrhagic. Sections of the walls of the abomasum and intestine showed engorgement of vessels and round-celled infiltration, but no bacteria. Sections of the hæmorrhagic and necrotic areas of the liver showed no bacteria in the former, but in the latter non-sporulating bacteria were found, particularly round the periphery of the lesion, where they were often present in considerable numbers. As these bacteria were not present in the hæmorrhagic areas, Dodd at first considered them to be mere secondary invaders, and did not regard them as the cause of the disease.

The absence of bacteria from the abomasal lesions established an important difference between this disease and that described by Jensen in 1915. In his later paper, Dodd surveyed the post-mortem examinations of 100 cases of black disease examined at the time of death. The almost constant presence of the characteristic necrotic lesions in the liver, two cases excepted, and the association therewith of a specific bacterium, compelled a modification of his earlier attitude regarding these bacteria. In considering the occurrence of the disease, the lesions and the experimental data, he reached the conclusion, which in its main still holds, that death was due to an acute toxæmia; that the toxæmia resulted from the absorption of toxin elaborated by bacteria in the liver lesions; that the bacteria

XUM

are confined to these lesions, except in rare instances, when the whole liver might show a mass invasion—two such cases recorded. Further, he incriminated the liver fluke with the appearance of the disease, considering it likely that this creature mechanically conveyed the bacteria from the intestine to the liver, and thus set up infection. The micro-organism regarded as the cause of the toxæmia was described in certain of its cultural characters, and the effect of its inoculation into sheep and laboratory animals was carefully recorded. It was not named, but that was of little consequence, and later workers on this disease have had no doubt of the identity of the bacillus described by Dodd. Black disease now appeared as an entity.

The observations made by Jensen in 1915 were confirmed by Gaiger[5] in 1922. Gaiger examined material sent to the laboratory from cases of braxy which had been slaughtered, or which had died. Material from twenty cases, mainly from the West of Scotland, was obtained in this fashion, and the only definite lesions appeared in the abomasum, and these corresponded to those described by Jensen. It now remained for Gaiger to identify the bacterium. He conducted post-mortem and bacteriological examinations on ten cases, all from the same district, of slaughtered animals, or animals which had just died. An anaerobic bacterium was recovered in each instance from the peritoneal fluid, the peritoneal surface of the abomasal lesions and the œdematous fluid of these lesions. The same bacterium appeared to be present in the blood stream and in the liver tissue whenever these were examined. The bacillus was identified as *V. septique*. Histologically the abomasal lesions in these ten cases again presented a picture comparable to that described by Jensen. The assumption was therefore made that the diseases in both cases were the same, and that the braxy or bradsot bacillus was *V. septique*.

An explanation of why or how the bacilli invaded the stomach wall has yet to be found. No experimental data were submitted by Gaiger on this point, but he sought to explain the phenomenon by accepting the hypothesis suggested by Nielsen—that the ingestion of frozen food and its presence in the lumen so chilled the walls of the adjacent abomasum that these were debilitated, thus facilitating bacterial invasion. But this is, indeed, a very questionable explanation. Again, Gaiger also considers changing conditions of climate and of food conducive to the appearance of the disease. Gaiger apparently felt that having identified *V. septique*, the question of the ætiology of braxy was settled, and likewise the oneness of braxy-like diseases. The suggestion in the Report of the Departmental Committee that more than one disease was included under the term braxy was regarded as erroneous. This conclusion may not be surprising in view of the lack of supporting evidence, but it is surprising to find a worker who has examined ten fresh cases writing in the following way about the views of a man who had critically examined and recorded his observations on over 100 cases in another part of the world. " The writer would not be surprised if Dodd, after further work, again altered his views. It may be that black disease in Australia will yet be shown

XUM

to be primarily a fourth stomach infection with *V. septique*, and that it is identical with braxy and bradsot." This confusion appears remarkable. However, Gaiger's enthusiasm for *V. septique* as the cause of disease, and his refusal to give unbiassed judgment to the evidence of another, will be best exemplified by quoting in full all his criticism of Dodd's work on black disease.

" Black disease occurs in New South Wales. It is said to be a ' braxy-like disease,' and it has been investigated by Dodd and others. Dodd's first article appeared in March, 1918. It was apparently published to show the negative cultural results he had obtained by dealing with live cases of black disease killed for the purpose of investigation.

" Miss Heller's criticism of Dodd's first article very aptly expresses the writer's views upon Dodd's findings. She says : ' I fear that Dodd's later investigation was undertaken to disprove an anaerobic ætiology of black disease. It does not seem to me that Dodd has at all disproved the possibility of anaerobic infection in his sheep cases. He has carefully avoided searching for his agent in the tissues most affected, the wall of the abomasum and of the intestine.' Further, discussing Dodd's sheep No. 34, Miss Heller remarks that ' Dodd had the truth in his fingers and failed to see it. The organism found in the original sheep killed a guinea-pig, 0.5 c.c. of the fluid from the guinea-pig killed a sheep with typical lesions of black disease, plus the typical local lesions of a vibrion septique infection; and because of the polymorphism of the organism, Dodd thought that several species were present, and that post-mortem invasion must have occurred. Dodd's bacteriologic criteria are weak throughout. So far as I can see, he calls the short forms ' braxy bacilli,' and the long ones malignant œdema organisms. There is no mention of any careful bacteriologic study in Dodd's paper.

" Dodd, in his second article, alters his previous views concerning the ætiology of black disease, and arrives at totally different conclusions. The liver lesion he describes as not constant, for he says : ' On two occasions where the animal showed clinical and post-mortem signs of black disease, no necrotic areas could be detected after search.' He arrived at the conclusion that ' the solution of the problem of the natural method of infection, and the situation of the primary lesion in the liver, is to be found in the liver fluke.' He believes that his bacillus lives in the sheep's intestine, and that cercariæ become contaminated there. He calls this evidence regarding the liver fluke ' circumstantial evidence.' His theory is evidently founded upon a belief that the cercariæ of the liver fluke arrive in the liver *via* the bile duct; but he would appear to be unaware of the work of Sinitzin, which shows that the cercariæ reach the liver by penetrating the wall of the alimentary canal and the peritoneal cavity, and reach the bile ducts by direct penetration of Glisson's capsule. If Dodd is aware of the liver fluke's true life history, then his theory of fluke carriers must be made to explain how the peritoneum does not become infected by the cercariæ *en route* to the liver.

" The writer would not be surprised if Dodd, after further work, again altered his views. It may be that black disease in Australia will yet be shown to be primarily a fourth stomach infection with the vibrious septique, and that it is identical with braxy and bradsot."

Gaiger overlooked the most fundamental portions of Dodd's work. Dodd as early as 1918 described the histo-pathology of the lesions in the abomasum, and remarked upon the absence of bacteria from these. This alone should have indicated that black disease differed from the bradsot and braxy lesions found by Jensen and Gaiger. Further, Dodd clearly showed that the most characteristic lesions were found in the liver, and not in the alimentary canal. Sheep 34 revealed a unique truth to Miss Heller and to Gaiger, which has never materialised. They considered that this animal died of *V. septique* infection, but there is no proof that this was so. However, even had it died from a *V. septique* infection it is surprising to find a veterinarian in 1922 conceiving that this organism could be the cause of black disease, when abundant evidence furnished by Dodd showed that it was not the cause of the disease. The two cases presenting atypical liver lesions were insignificant when compared with the overwhelming number, more than ninety-eight cases, showing typical liver lesions. Furthermore, although Dodd's conception of the life history of the liver fluke may have been faulty, his

XUM

original observations concerning its ætiological significance are generally accepted to-day.

Confirmation of Dodd's work comes from Albison[1] (1927) in Victoria, who described black disease, which he named infectious necrotic hepatitis. Albison recognised the similarity of the causal micro-organism, particularly of its toxin, to *B. œdematiens*. The same year Turner and Davesnes[19] described the black disease bacillus in detail, and though noting its greater size and slower growth, they regarded it as a variant of *B. œdematiens*.

In Germany, Zeissler and Rassfeld[22] (1927) about the same time isolated from the carcases of bradsot sheep a bacillus they called *B. gigas*, and which resembled *B. œdematiens* closely in many of its growth characters. Prior, aparently, to Zeissler and Rassfeld's work, Meyn[16] had isolated *B. œdematiens* from bradsot carcases from Iceland, and in 1927, Miessner and Meyn[14] reported the isolation of *B. œdematiens* from bradsot carcases, and suggested that the peracute form of bradsot might be attributable to the action of the toxin of that bacillus. Evidence in 1927 was accumulating to show that *B. œdematiens*, or a similar bacillus, must be reckoned with in a consideration of bradsot in Europe.

The important rôle played by *B. œdematiens* in black disease was already apparent, and was shortly to be more fully emphasised. Edgar[4] (1928), by cultivating from large quantities of liver tissue from normal sheep from black disease districts, had in a number of instances isolated the black disease bacillus. He failed to find the organism in the liver of sheep from districts where black disease was unknown. This cultivation of the causal organism from the livers of normal sheep has been confirmed by Turner.[21]

Turner[20] (1928) showed that the spores of *B. œdematiens* may lie latent in the tissues of rabbits, and later he demonstrated that these spores were innocuous for sheep when inoculated into the body by various routes, or when they reached the body tissues after ingestion, the spore apparently having passed the mucosal barrier of the alimentary canal. These spores were conserved in various parts of the body, particularly the liver, spleen and bone marrow, where they might remain viable, but quiescent for periods of several months. Spores being latent in the liver, it remained to find some factor which would overcome any inhibition to germination, thereby permitting metamorphosis to vegetative bacilli, the production of toxin and of disease. It is now considered that the immature liver fluke in traversing the liver tissue causes cell injury and necrosis, thereby giving rise to suitable nidi for the germination of latent bacterial spores, should these be present in the tissues, and that thus the disease with its characteristic liver lesions is brought about. We have seen that black disease occurs in New South Wales and Victoria. Hopkirk[6] has reported it in New Zealand, and it probably occasions losses in Tasmania. The ætiology may be considered fairly well established.

The disease described by Miessner in 1909, in Germany, resembled black disease, and in 1931, Miessner, Meyn and Schoop[15] returned to a consideration of what

XUM

they call the German bradsot. From their description the lesions of this disease correspond most closely with the lesions of black disease, and the causal organism of the German bradsot found in the necrotic liver lesions is described and named *B. gigas*, a bacillus toxicologically and serologically indistinguishable from *B. œdematiens*, and in all probability identical with the causal organism of black disease. These authors consider the German bradsot is practically identical with black disease, but there is not such constant evidence of recent fluke infestation, and the rôle played by the liver fluke, if any, is not clear. The disease occurs amongst animals grazing on low-lying pasture which is subject to inundation, and at a time of year, from late summer to winter, when fluke invasion might be expected; but it also appears in animals which are stall fed with hay from the incriminated land, and it would be difficult to suspect the liver fluke under these latter circumstances. The authors, however, remark upon the difficulty of obtaining fresh material, and only give details of five post-mortem examinations, two of which were not on the freshest of carcases. It would seem that they had erected much of their theory regarding the occurrence of the disease upon hearsay and not upon personal knowledge and examination of cases. Nevertheless, the evidence furnished by the article, particularly when considered along with the findings of Zeissler and Rassfeld, Miessner and Meyn, and Miessner, must be regarded as showing that *B. œdematiens* is capable of causing bradsot in Europe.

The same bacillus has been recovered by McEwen[10] from the tissues of sheep which had died of braxy-like disease in this country. The material was examined, sometime after death, upon its receipt at the laboratory. From four specimens received from the hilly districts of Wales, the bacillus was isolated on three occasions. It was isolated in one instance only from a very large number of examinations on material from the Romney Marsh district. From these experiences the author felt that the bacillus, though apparently of little importance on the Romney Marsh, might yet be shown to have an ætiological significance in the sheep diseases of other parts of the country.

Briefly recapitulated, black disease regarded at one time as a form of braxy, has now been shown to be due to an infection with a bacillus of the *B. œdematiens* type, and data have accumulated to show that the same bacillus causes a similar disease in Europe. Braxy or bradsot was formerly attributed to a special braxy or bradsot bacillus, but its identity was not clear, and until comparatively recent times various bacilli probably masqueraded under these names. In 1922, *V. septique* was incriminated as one cause of braxy. There are therefore two micro-organisms which may cause the braxy-like diseases of Europe: *V. septique* and *B. œdematiens*, or *B. gigas*.

A brief consideration of these micro-organisms will show that *V. septique* is a highly pathogenic bacillus most tenacious of its pathogenicity. It does not readily become avirulent in artificial culture; it is a most vigorous grower; it is not very exacting in its anaerobic requirements; parenteral inoculation when fatal, and it is seldom otherwise when whole culture media or tissue fluids are

XUM

inoculated, even though the inoculum be small, results in a generalised blood-stream infection as well as an extensive local lesion. The bacilli are easily recovered from the blood and organs of the inoculated animal. In contra-distinction to these strong positive qualities, we find that *B. gigas*, though it may be highly pathogenic when isolated, may readily lose the capacity to kill laboratory animals (personal observation). It is most exacting in its growth requirements, demanding a very complete anaerobiasis. Parenteral inoculation seldom results in a blood stream or generalised infection, usually the bacilli are only recoverable from the neighbourhood of the inoculation lesion. If *V. septique* and *B. gigas* or *B. œdematiens* were mixed in varying proportions and inoculated into animals, there is little doubt that *V. septique* would invariably be recovered, the other would be readily lost.

Bearing the characteristics of the two species in mind, it is instructive to refer back to Nielsen's findings (1897), and to the Report of the Royal Commission in 1906. They had great difficulty in producing fatal disease in sheep by inoculations of bradsot and braxy material. The same material inoculated into guinea-pigs gave very inconsistent results. Nielsen clearly states that the bacilli were often recoverable from the local lesion, but not from other body tissues. Further, he remarks upon the marked loss of pathogenicity. Had Nielsen's bradsot bacillus been the *V. septique*, surely he would not have had such results. The results are, however, more compatible with the conception that a *B. œdematiens* type or a comparatively non-pathogenic bacillus was being worked with.

Nevertheless, there would be nothing surprising if these workers had occasionally encountered *V. septique*, even assuming for the moment that it was not the cause of the diseased conditions they were investigating. McEwen and Roberts[11] (1931) showed that *V. septique* may be present in carcases of sheep when there is evidence that death had been caused from infection with an entirely different anaerobic bacillus. There was good reason to regard it as a post-mortem invader. Therefore it would be neither surprising nor significant if *V. septique* had been occasionally found in cultures handled by Nielsen and others who worked with old post-mortem material, or, as in Jensen's case, putrid blood, pickled stomach, and dried smears on glass slide. Moreover, it would have been very surprising if they had failed to recover such a highly pathogenic anaerobe as *V. septique* had this been the usual cause of the diseases they investigated. It seems obvious that *V. septique* was frequently absent from their material, and that in many instances the evidence is all against this micro-organism having been the cause of disease in the cases investigated.

Before closing the consideration of braxy-like diseases, attention may be called to the disease of sheep on the Romney Marsh described by McEwen and Roberts[11] (1931). This is attributed to a bacillus named *B. paludis* (McEwen,[9] 1930), which morphologically and culturally is indistinguishable from *B. welchii*, although it produces a more potent toxin not neutralised by *B. welchii* antitoxin. The lesions of the disease are characteristic, consisting of an enteritis, often acute, and

XUM

accompanied by ulceration; and a peritonitis, where the peritoneal effusion is most abundant, with marked injection of the peritoneal vessels, giving to the peritoneum the appearance of red meshed netting. This disease may occur without evidence of tissue invasion by the bacilli, though invasion often occurs. It is suggested that toxin is produced by *B. paludis* growing in a pabulum of the contents of the intestine, that this toxin injures the mucosa, enters the body, causing toxæmia and death. The evidence to support the suggestion is the demonstration of the toxin in the contents of the intestine, and the fact that the disease may be reproduced with the characteristic lesions by feeding large quantities of the bacteria in broth culture or suspended in fresh nutrient. Feeding bacteria suspended in normal saline does not produce the disease. The disease is, therefore, experimentally produced when the bacteria are introduced into the alimentary canal along with abundant material suitable for growth and toxin elaboration, but not when they are present in the alimentary canal, deprived of abundant material for growth requirements. The bearing of these facts upon the occurrence of the disease in the field is not yet clear, and requires further investigation; but as concerns our present discussion, it is of interest to emphasise that a braxy-like disease is caused by an anaerobic bacterium, neither *V. septique* nor *B. œdematiens*, which launches its attack upon the body from the alimentary canal.

" Each is caused by a specific anaerobe which most likely in the whole of them gains entrance to the body through the stomach and intestines."[18]

[Since this article was sent to the press, *Bulletin No.* 57, *Council of Scientific and Industrial Research, Commonwealth of Australia*, has been received. This Bulletin is written by Dr. H. W. Bennetts, and is entitled " Infectious Enterotoxæmia of Sheep in Western Australia." It describes a disease of sheep caused by an anaerobic bacillus of the *B. welchii* type, named *B. ovitoxicus*. The disease is considered to be an acute toxæmia of sheep following the rapid multiplication of the micro-organisms within the contents of the small intestine, and it would appear to have much in common with the disease described by McEwen and Roberts (1931).]

REFERENCES.


[1] Albiston, H. E. (1927): *Aust. J. Exp. Biol. Med. Sc.*, 4, 113.
[2] Dodd, S. (1918): *J. Comp. Path. and Therap.*, 31, 1.
[3] Dodd, S. (1921): *J. Comp. Path. and Therap.*, 34, 1.
[4] Edgar, G. (1928): *Aust. Vet. J.*, 4, 133.
[5] Gaiger, S. H. (1922): *J. Comp. Path. and Therap.*, 35, 191 and 235.
[6] Hopkirk, C. S. M. (1927): *New Zea. J. Agric.*, 35, 141.
[7] Jensen, C. O. (1896): *Deuts. Z. Theirmed.*, 4, 249.
[8] Jensen, C. O. (1915): *Z. Infekt. Kr.*, 17, 1.
[9] McEwen, A. D. (1930): *J. Comp. Path. and Therap.*, 43, 1.
[10] McEwen, A. D. (1931): *J. Comp. Path. and Therap.*, 44, 149.
[11] McEwen, A. D., and Roberts, R. S. (1931): *J. Comp. Path. and Therap.*, 44, 26.
[12] Miessner, H. (1908): *Berl. Tierärztl. Wschr.*, 577.
[13] Miessner, H. (1909): Ders., Mitteilungen des Kaiser-Wilhelm-Institut Landwirtschaft Bromberg (not seen in original).
[14] Miessner, H., and Meyn, A. (1927): *Deuts. Tierärzth. Wschr.*, 34, 548.
[15] Miessner, H., Meyn, A., and Schoop, G. (1931): *Zbl. Bakt. Abt.*, 1, *Orig.*, 120, 257.
[16] Meyn, A., reported by Miessner, H., and Meyn, A. (1927): *Deuts. Tierärztl. Wschr.*, 34, 548.


XUM

[17] Nielsen, I. (1897): *Mhft. prakt. Zierheilk.*, 8, 55.
[18] Rept. Dept. Comm. on Louping Ill and Braxy, 1906.
[19] Turner, A. W., and Davesne, J. (1927): *C. R. Soc. Biol.*, 96, 921.
[20] Turner, A. W. (1928): *C. R. Soc. Biol.*, 98, 558.
[21] Turner, A. W. (1930): Aust. Council for Scientific and Industrial Research, Bull. 46.
[22] Zeissler, J., and Rassfeld, L. (1929): *Arch. wiss. prakt. Tierhlk.*, 59, 415.

# SOME EXPERIMENTS IN RINDERPEST VACCINATION: ACTIVE IMMUNISATION OF INDIAN PLAINS CATTLE BY INOCULATION WITH GOAT-ADAPTED VIRUS ALONE IN FIELD CONDITIONS.

By Major R. F. STIRLING, I.V.S.

*Director of Veterinary Services, Central Provinces, India.*

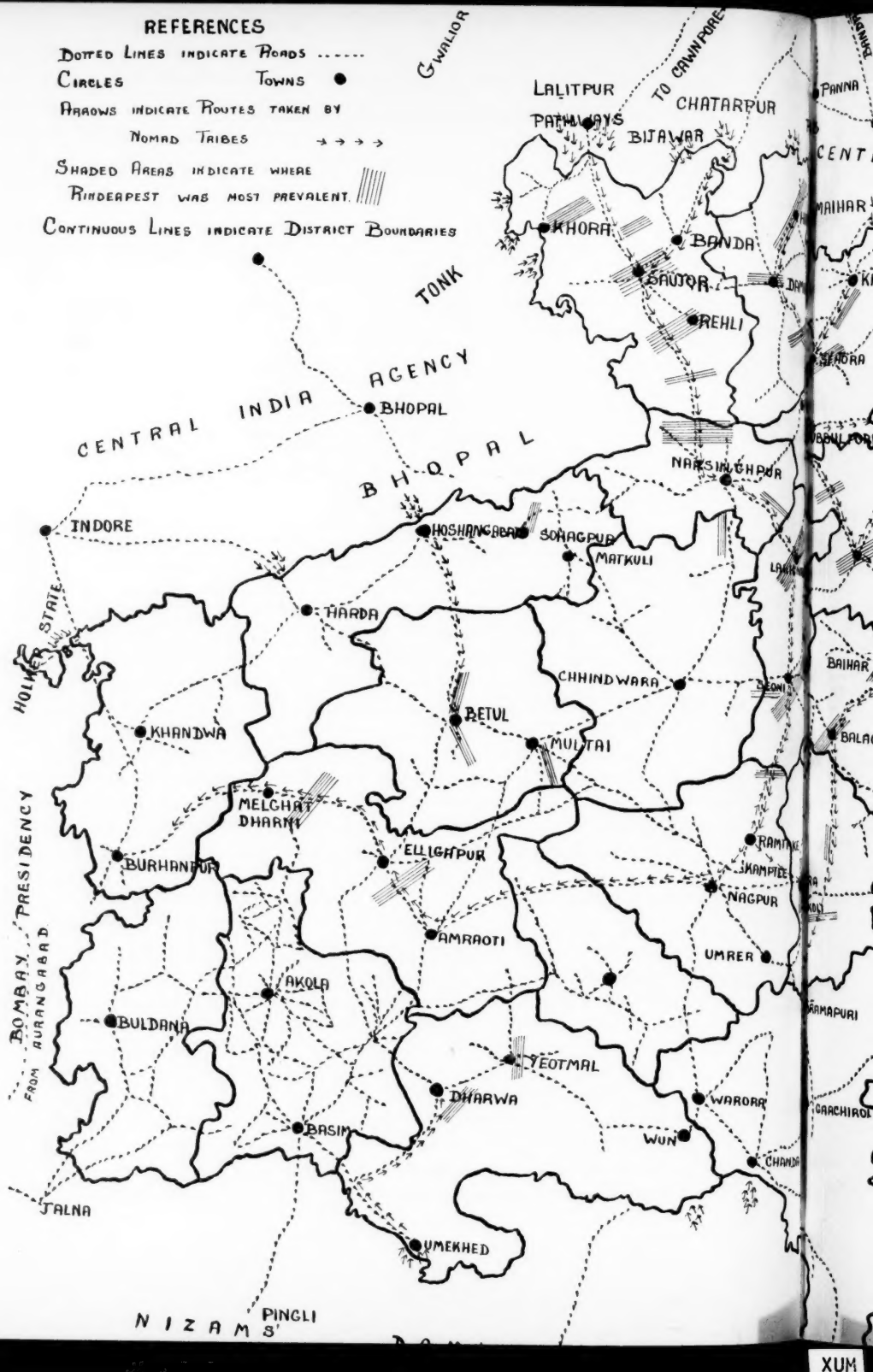
## I. Introduction.

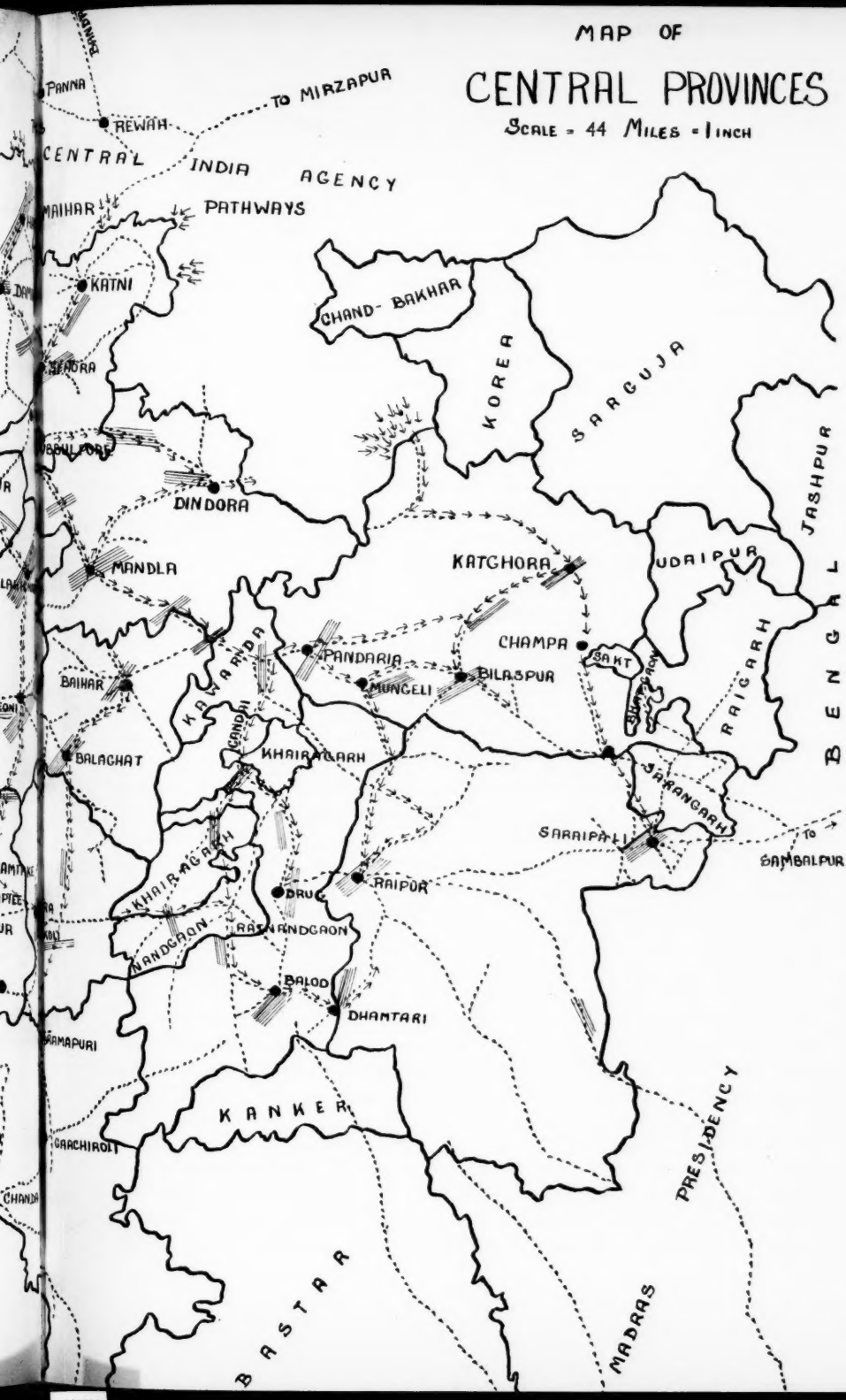
RINDERPEST in the Central Provinces presents a problem which differs perhaps somewhat from that of the disease elsewhere in India. The province is land-locked, is surrounded for the greater part by Indian states, and also contains small states within its confines. Even when there exists any veterinary organisation in these states, it is not comparable with that of British India. In addition, the province is dependent to a large extent upon these states for an adequate supply of cattle for its agricultural needs. About 100,000 cattle are thus imported each year. A scheme has been prepared by the veterinary department of the province whereby a number of inspection and quarantine camps are to be placed within its borders, and imported cattle admitted only through such stations. By the imposition of a small duty, these stations would become in time self-supporting. At the present moment this proposal is under the careful consideration of the local Government, but they may feel that the initial expenditure is not justifiable in these days of financial stringency; there is also the further question as to whether the capitation tax might not restrict importation to such a degree that the farmers of the province would be embarrassed for want of cattle.

The importation of cattle into the province is attended with danger of two kinds. The first kind, which is an obvious one, is that of introducing disease, especially rinderpest, to which danger the attention of the veterinary department is chiefly directed. The second kind of danger is the more serious one. The imported cattle come mainly from the north and north-west of the province, and their main destination is the south-east. The distance travelled by road may be anything up to 400 miles. From September till March, hordes of cattle move along the main roads of the province, halting at or near villages. If these animals are not diseased when they enter the province (which is most likely to be the case, as they are imported for sale) they certainly often contract the disease after entry, and carry it from place to place. Perhaps the worst feature of this trade is that

LIBRA
JUN 4

XUM


REFERENCES
Dotted Lines indicate Roads .....
Circles Towns ●
Arrows indicate Routes taken by Nomad Tribes → → → →
Shaded Areas indicate where Rinderpest was most prevalent.
Continuous Lines indicate District Boundaries
GWALIOR
TO CAWNPORE
LALITPUR
PATHLAYS
CHATARPUR
BIJAWAR
KHORA
BANDA
GAUJOR
REHLI
TONK
CENTRAL INDIA AGENCY
BHOPAL
BHOPAL
NARSINGHPUR
INDORE
HOSHANGBAD
SOHAGPUR
MATKULI
HOLKAR STATE
HARDA
CHHINDWARA
BETUL
KHANDWA
MULTAI
MELGHAT
DHARNI
ELLICHPUR
BURHANPUR
RAMTEK
KAMPTEE
NAGPUR
AMRAOTI
UMRER
AKOLA
BULDANA
BOMBAY PRESIDENCY
FROM AURANGABAD
YEOTMAL
DHARWA
WARORA
WUN
BASIM
JALNA
UMEKHED
NIZAM'S
PINGLI
PANNA
MAIHAR
BAIHAR
BALAG
RAMAPURI
GARCHIROL


MAP OF
CENTRAL PROVINCES
SCALE = 44 MILES = 1 INCH
PANNA
REWAH
TO MIRZAPUR
CENTRAL INDIA AGENCY
MAIHAR
PATHWAYS
KATNI
CHAND- BAKHAR
KOREA
SARGUJA
JASHPUR
BENGAL
DINDORA
MANDLA
KATGHORA
UDAIPUR
CHAMPA
SAKT
PANDARIA
MUNGELI
BILASPUR
BAIHAR
KAWARDHA
RAIGARH
BALACHAT
KHAIRAGARH
SARAIPALI
SAMBALPUR
DRUG
RAIPUR
RAJNANDGAON
BALOD
DHAMTARI
KANKER
BASTAR
MADRAS PRESIDENCY

XUM

the dealers sell at the roadside villages any cattle which seem to them to be slightly ailing, or if the cattle are too ill to deceive the villagers they are turned loose at places where the dealers halt at night, that is, in the neighbourhood of a village.

In 1929, I attached a map to the Annual Report of the veterinary department, showing the routes taken by the nomadic dealers. This map, which is reproduced here, demonstrates how the incidence of cattle disease in the province follows these routes.

For many years the veterinary department has been combating outbreaks of rinderpest by means of the serum-alone method of inoculation. In the year 1928–1929, 171,153; in 1929–1930, 251,088; and in 1930–1931, 115,824 cattle were thus inoculated, at a cost of roughly £4,000 a year. This sum may seem small when judged by European standards, but when it is realised that the total expenditure of the veterinary department is only about £30,000 annually, the portion expended upon serum for the above inoculation is a large one. Moreover, the limitations of this method of protection, which confers a passive immunity usually lasting no longer than nine days, are now so well known that they need not be elaborated here.

In the year 1930–1931, active immunisation by means of the serum-simultaneous method of inoculation was adopted, and over 12,000 animals were inoculated by the method. The work done by Dr. J. T. Edwards when he was Director of the Imperial Institute of Veterinary Research, Muktesar, had made this method much more easy of performance in the field than was possible formerly. The virus used, according to Dr. Edwards's method, was the "fixed" goat virus, obtained from the Muktesar Institute. This was maintained alive locally by passage through susceptible goats. The anti-rinderpest serum employed for inoculation, simultaneously with the virus, of the cattle to be immunised, was the specially potent serum ("Class II serum") obtained from the same Institute. The operations were placed in charge of a senior officer of the department, and the intention was that he should visit all districts in the province in turn, and train two or three assistant veterinary surgeons in each district to do the work.

In a few months it was found that the departmental budget could not bear the strain of the extra expenditure incurred in these operations. The main item of extra expenditure, apart from the cost of staff, was the cost of the serum prescribed for the inoculations. The serum itself came to over two shillings a head with most of the animals inoculated. This sum may seem trifling again, but it must be remembered that there are twelve million cattle in the province, and that there is some evidence that the duration of the immunity conferred by the method may be sometimes under three years (Edwards, 1927)[1]. Even if only one-half of the cattle in the province were immunised by this method—and I think the other half may be taken as probably not worth protecting—the outlay would come to £60,000, or perhaps an expenditure spread over a number of years of about £20,000 annually. This expenditure could not be met within the veterinary budget of the province even in times of plenty. Unfortunately,



XUM

the operations started in 1931 coincided with the universal financial depression, and so had to be discontinued for want of money.

As has been said, the chief obstacle to the widespread performance of the serum-simultaneous inoculations was the financial one, represented by the cost of the serum, and the question now remained whether the average dose of serum inoculated could be safely reduced, or, indeed, eliminated altogether. There seemed warranted a considerable degree of expectancy in this direction.

In the bulletin written by Edwards[2] (1930), in which he described the adaptation of the virus of rinderpest to goats, the decrease in virulence of the virus for hill cattle after prolonged passage through goats, and the possibility of using this attenuated virus for sure, safe, and easy active immunisation by the serum-simultaneous method on a large scale, the following sentences had been noted: " Early in the course of this year [1928], three plains young cattle were inoculated as controls by one of my colleagues (Mr. M. B. Menon) on an establishment (Karnal), where numbers of cattle were being subjected to the serum-simultaneous inoculation with the use of goat virus. It was very noteworthy that all these controls survived with hardly any visible impairment in health, although their temperature charts showed that they had passed through a distinct rinderpest reaction " (*p.* 10). . . .

" It has already been explained that the ' goat virus ' is one that has now become much attenuated in comparison at least with the unmodified cattle virus. The attenuation has reached the stage in several of our tests when fortification of the animal against it by simultaneous inoculation of serum is unnecessary, even when the tests have been undertaken upon such highly susceptible races of cattle as typical Himalayan hill bulls; we may presume, *a fortiori*, that fortification would be still more unnecessary with the ordinary plains races of cattle in India " (*p.* 15).

A discussion with the officer in charge of serum-simultaneous operations with reference to the first passage quoted above and to our local experience with controls, led the officer (Mr. P. S. Nair) to relate to me an experience which he had had while I was on long leave out of India, and of which I had consequently no previous knowledge. I will quote his words: " Whilst carrying out serum-simultaneous inoculations at Mangrulpir, five cattle (two cows and three buffaloes) were inoculated with goat virus, and then it was found that all the serum was expended. These animals showed a staring coat, discharge from the eyes, great thirst, and lack of appetite. They all eventually recovered. Unfortunately, no temperature charts could be maintained." Here were, therefore, two records of plains cattle having recovered from injections of the virus alone. Would this non-fatal attack which followed inoculation with the goat virus produce sufficient immunity to enable these plains cattle to withstand a dose of highly-virulent hill bull virus, which, given alone, would undoubtedly cause many fatalities? This was the first question faced, and to which it was desired early to obtain an answer.

XUM

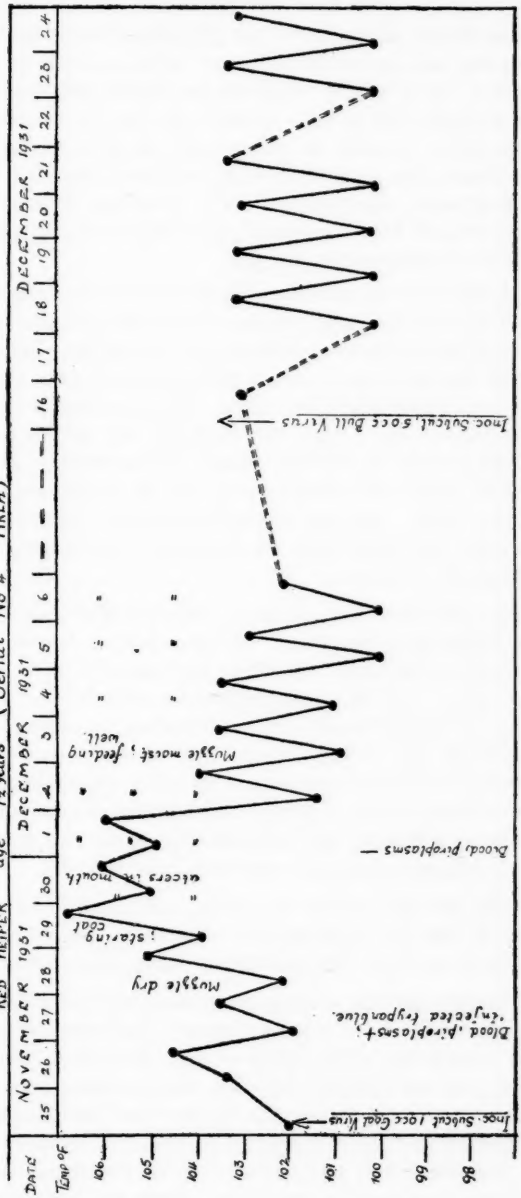



CHART No. 1.

Cattle (Batch No. 1) inoculated with goat virus; immunity tested later, in 21 days, by inoculation of hill bull (Muktesar) virus.

## II. Experimental.

As there was no budget allotment for the purchase of cattle for experimental purposes, the first step that had to be taken was to find a cattle owner who was prepared to take the risk of having his cattle inoculated with goat virus alone. At the time this risk was felt to be a distinct one, for the evidence available concerning the complete recovery of plains cattle after the virus inoculation referred to eight animals only, namely, the three at Karnal, described by Edwards, and the five at Mangrulpir, mentioned above. It was not impossible that the recoveries in these animals might be due to some degree of immunity resulting from a slight attack of rinderpest in the past.

At this time (April, 1931) the veterinary department had been helping in a rural uplift movement in a group of villages, and one of the cattle owners of a village lent some cattle to the department for experiment, after he had been induced to make a written statement that the experiments would be carried out at his risk. Four animals were inoculated with goat virus alone. The reactions produced in these animals were not very severe. After the reactions had subsided each animal was inoculated with a dose of hill bull virus; all survived. The above preliminary experiment, which was carried out by Mr. M. James, encouraged us to proceed with further work. An area was selected in the Chhattisgarh division of the province, where the local cattle are numerous, very cheap, and of small size, mostly under 300 lb. in weight.

*Technique.*—(1) Goat virus was obtained from the Muktesar Institute and injected at once locally into two goats. When a definite febrile reaction was registered, they were bled for virus. (2) This virus was injected in doses of 1 c.c. into a number of cattle. (3) At the same time the virus was injected into two more healthy goats to control the activity and maintain the supply of the virus. (4) The reactions in (2) and (3) were noted. (5) When the reactions produced by the inoculation of goat virus had completely subsided, usually in about eighteen days after the inoculation, a dose of cattle virus (hill bull virus obtained from the Muktesar Institute) was injected into all animals. Again two goats were kept as controls. (6) Subsequent reactions were noted and recorded.

*Experiments.*—In all, ten batches of cattle have been inoculated by this double virus method, and the total number of cattle inoculated at the time of writing has amounted to 343. The results are summarised below:

*Batch No.*1.—Eleven animals, varying in age from one and a-half to fourteen years, in fairly good condition. From local inquiry, rinderpest had not occurred in the village for some years. The supply of goat virus from Muktesar, in the form of the mixed contents of three ampoules, was inoculated, according to the accompanying directions, into two healthy goats that had shown a constant pre-injection temperature. One of the goats supplied the required virus on the fifth day after injection. The temperatures of all experimental cattle were normal before the inoculation of the goat virus. Each was inoculated, according

XUM

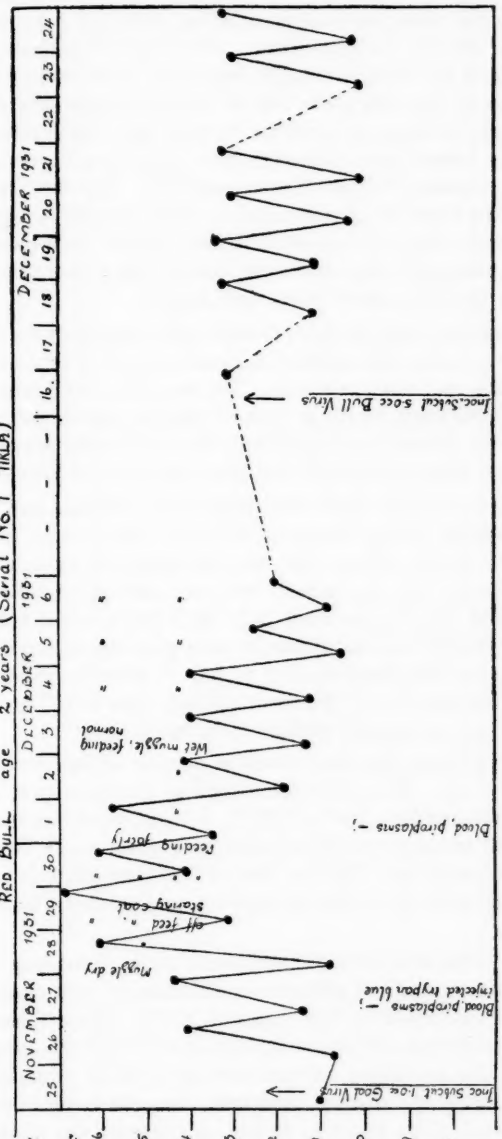



CHART No. 2.

Cattle (Batch No. 1) inoculated with goat virus; immunity tested later, in 21 days, by inoculation of hill bull (Muktesar) virus.

to the technique already described, with 1 c.c. of the goat blood subcutaneously in the neck, with the usual precautions regarding sterility; 5 c.c. was inoculated at the same time into two healthy goats. All inoculated animals showed a rise of temperature from the third to the fifth day after inoculation. A staring coat and dry muzzle were the only other clinical symptoms generally noted; in one animal a slight mouth lesion was seen on the fifth day; none refused food completely, and none showed any alimentary disturbance (*see* Charts Nos. 1 and 2, for record of the reactions in two of these animals). The fæces were examined for coccidia and the blood for piroplasms as a routine measure; should any show numbers of coccidia, these animals were to be excluded from experiment; any that showed piroplasms (*B. bigemina*) were treated with trypanblue. The rinderpest reactions in the two control goats were distinct.

The test inoculation with cattle virus was made with Muktesar hill bull virus. Seven of the above cattle were injected, each with a dose of 5 c.c., on the twenty-first day after the goat virus injection. The remaining four cattle were not inoculated, but kept in reserve for the purpose of a further experiment, to be described later. None of the animals inoculated with the cattle virus showed any distinct thermal or clinical reaction, and are still alive and well (*see* Charts Nos. 1 and 2).

*Batch No.* 2.—Forty-four cattle, including three buffaloes and three bullocks of the more susceptible breeds, composed this herd; ages varied from six months to twelve years. In this village there was no history of rinderpest for at least four years previously. As the animals were not suitably stabled and tethered, the reactions could only be recorded daily in a few selected animals. It was noticed that the younger animals showed a more pronounced thermal and clinical reaction (temperature ranging from 104° to 108° F. after the third day) than the older animals. The test dose of Muktesar hill bull virus was given to only thirty-one of this herd; all successfully withstood the inoculation.

*Batch No.* 3.—Fifteen cows and calves in another village were inoculated as above with goat virus. The rinderpest reactions produced were quite distinct, the temperature ranging from 104° to 106° F. Some of the calves went temporarily off their feed, but subsequently all animals became normal. The goat controls also showed good reactions. The test dose of Muktesar hill bull virus was given seventeen days after the goat virus inoculation, and produced hardly any noticeable disturbance.

*Batch No.* 4.—One hundred and twenty-one cattle, consisting of 45 bullocks, 31 cows, 36 calves, and nine buffaloes were inoculated with goat virus. There was a history of rinderpest having occurred in this village (Chataud) in 1931, but the two cases reported were sporadic, the rest of the animals escaping infection. The reactions in the inoculated animals were again more pronounced in calves, but all showed clinical and thermal reactions. One old bullock developed severe coccidiosis and died. The reactions in the control goat was distinct. The test inoculations were carried out with virus obtained from local bull calves, inoculated with cattle virus, and showing distinct reactions. All animals withstood the test.

XUM

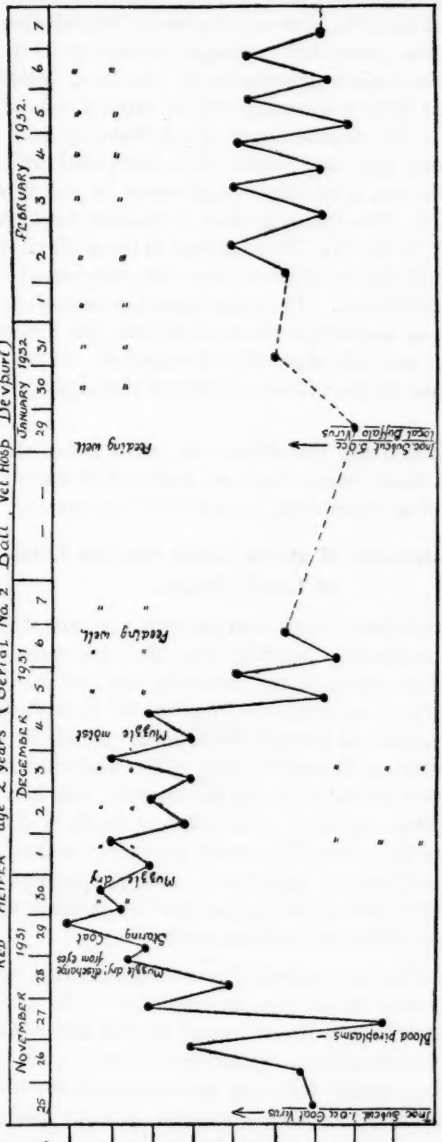



CHART No. 3.

* Cattle (Batch No. 1) inoculated with goat virus; immunity tested later, in 65 days, by inoculation of cattle virus obtained locally from a natural outbreak.

Charts Nos. 5 and 6 show reactions in control cattle inoculated with the cattle virus only.

*Batch No.* 5.—The technique was modified in this experiment by using instead of goat virus obtained from the Muktesar Institute, hill bull virus obtained from that institute, which was successfully passaged through goats in the field. This seemed to produce the desired attenuation in virulence, besides cleansing the virus of the piroplasms liable to contaminate it in cattle blood. The modification was adopted owing to the increased cost of the Muktesar goat virus. Twenty cattle, comprising cows and their calves, were inoculated with the goat virus thus obtained. There was a history of rinderpest in the village (Nawagaon) three years previously. The calves showed a thermal reaction from the fifth day ranging from 103° to 104° F. The reactions in the adult animals were almost negligible ; this may be due to recovery from the outbreak of rinderpest which prevailed three years previously. The control goats inoculated, as in the preceding experiments, showed an undoubted thermal reaction, the temperature rising to 105° F. on the fourth and fifth days after inoculation. It was not possible to perform the further test by inoculation of hill bull virus upon the animals of this batch.

*Batches Nos.* 6, 7, 8, 9 and 10.—These comprised 100 cattle, and nothing of interest was noted in them, except that one buffalo calf showed a very marked reaction to the goat virus inoculation, from which it recovered.

### Test of Immunity of above Cattle against Cattle Virus of Local Origin.

In Batch No. 1, described above, four animals were not inoculated with hill bull virus, but were reserved in the hope that their immunity might be tested by means of cattle virus obtained from naturally infected cattle in the scene of an outbreak of rinderpest occurring at a convenient distance from the village. Such an outbreak was seen on January 27th, 1932. Batch No. 1 had been inoculated with goat virus on November 25th, 1931. Unfortunately, only two out of the four animals were available for the further test ; one had been sold out of the district, and another had died. The cause of death in this animal cannot be definitely traced, as the owner did not seek veterinary assistance, but he states that there was no evidence of rinderpest ; this statement may be accepted without reserve, for the Indian peasant is only too familiar with the slightest symptom of the disease which he dreads so much.

In Charts Nos. 3 and 4, the reactions of the two animals that were now available for test to the goat virus inoculation are recorded. The operator in charge, moreover, records that the reaction displayed by the heifer (Chart No. 3) was much more marked than that shown by the cow (Chart No. 4). The charts also show the temperature reactions following upon inoculation with the local cattle virus. From the natural outbreak of rinderpest, virulent blood was taken from a buffalo calf suffering from the clinical disease, with a temperature of 106° F. The outbreak in the village in which this calf was selected was a normal one for this part of India, inasmuch as sixty-three animals died out of ninety-eight attacked.

XUM

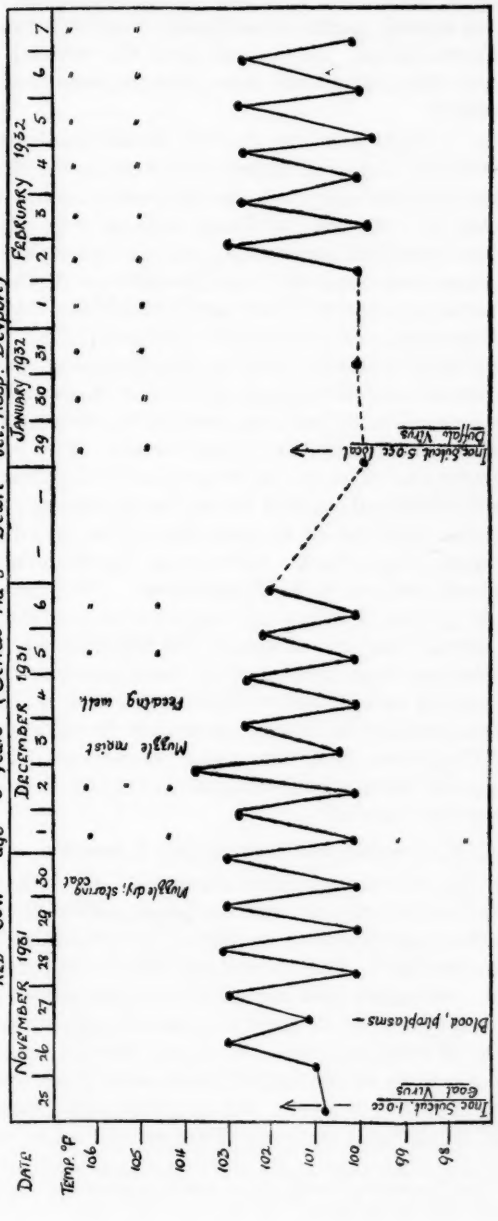

No. 4
Red Cow age 6 years (Serial No. 6 Bewri Vet. Hosp. Devpuri)
Date
Temp. °F
106
105
104
103
102
101
100
99
98
25 | 26 | 27 | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 | — | — | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7
November 1931
December 1931
January 1932
February 1932
Muggle dry: starring coat
Muggle moist
Feeding well
Blood, piroplasms
Tissue Sucket, 1·0 cc Goat Virus
Tissue Sucket 5·0 cc Goat Buffalo Virus

CHART No. 4.

Cattle (Batch No. 1) inoculated with goat virus; immunity tested later, in 65 days, by inoculation of cattle virus obtained locally from a natural outbreak.

Charts Nos. 5 and 6 show reactions in control cattle inoculated with the cattle virus only.

The two animals of Batch No. 1, already mentioned, namely (1) a heifer, and (2) a cow in the seventh month of pregnancy, were each inoculated with 5 c.c. of the above buffalo blood. At the same time, the virulence of the blood was controlled by inoculation of two bull calves with the same dose of the blood (*see* Charts Nos. 5 and 6).

From the charts it will be seen that the two animals previously inoculated with goat virus showed no febrile rise in temperature during the time their temperatures were taken, *i.e.*, until the ninth day after inoculation with the cattle virus; both animals fed well, and no clinical changes were observed. The officer in charge of the operations states: "at each time when the temperatures were taken in these experimental animals I made it a point to take the temperature of a few of the cattle grazing along with them, and I am satisfied that they showed almost the same temperature, with a variation of 0.2 and 0.6° F.°" Experience shows that the temperature of normal cattle in India is erratic, possible owing to the absence of constant supplies of drinking water in the grazing areas. The reactions in the two control calves are thus described by the operator: "Both the bull calves used as controls showed a typical reaction. They passed through all the stages of rinderpest. The rise in temperature in both began from the fourth day, followed by impaired appetite, dry muffle, staring coat, and discharge from the eyes. Vesicles were seen on the gums and tongue after the eighth and ninth days. The fæces of both became soft between the ninth and tenth days. At this stage the animals were sent to the slaughterhouse. Post-mortem examination revealed typical patches of ulcers and congestion of the intestines." The reason why these animals were not allowed to die naturally was because they were gifts to the veterinary department, and, in consequence, salvage had to be permitted. Both animals were almost moribund at the time of slaughter. The feelings of relief at the results obtained are expressed at the end of my colleague's interim report: "*Conclusion*—goat virus alone vaccination confers immunity against rinderpest, as the animals have withstood a massive dose of local virus after a lapse of over two months."

### III. Discussion and Concluding Remarks.

It will be seen that with the exception of one old bullock that died as the result of resuscitation in virulency of latent coccidiosis, all the 343 Indian plains cattle subjected in these experiments to inoculation with rinderpest virus adapted to goats by passage survived.* They showed undoubted mild rinderpest, but no serious disturbance. On testing their immunity later with cattle virus, either of Muktesar hill bull origin, or from the scene of a natural outbreak in the vicinity, they were found to be fully protected. It would, therefore, seem that cattle belonging to these races can be successfully immunised, without unduly serious risk, by inoculation of goat virus alone; and simultaneous inoculation of serum, as is prescribed in the ordinary serum-simultaneous method of immunisation,

*At the time of despatch of this paper for publication (March 2nd, 1932), one of the operators-in-charge (Mr. G. D. Pani) has reported briefly the successful inoculation of over 200 more cattle with locally adapted goat virus without fatality.—R. F. S.

XUM

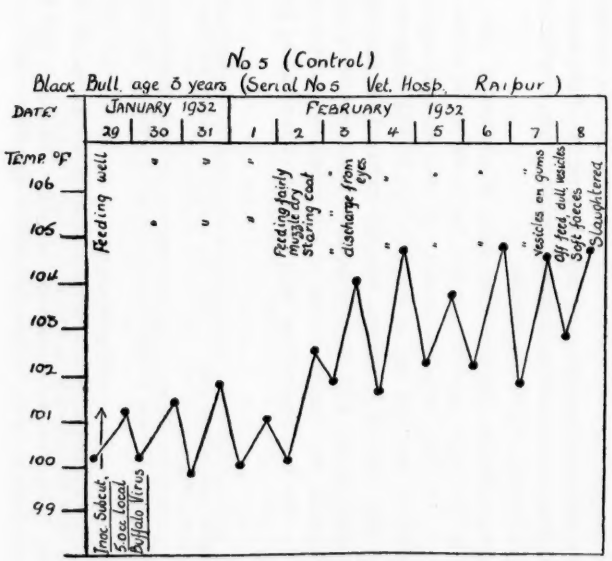



CHART NO. 5.

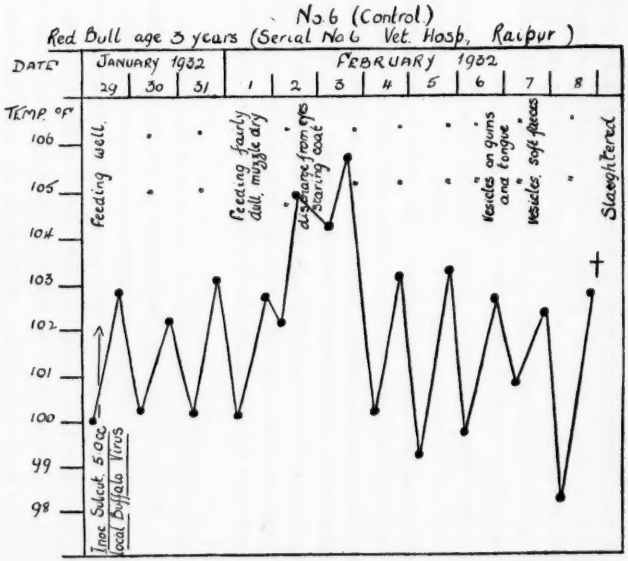



CHART NO. 6.

Cattle (Batch No. 1) inoculated with goat virus; immunity tested later, in 65 days, by inoculation of cattle virus obtained locally from a natural outbreak.

These charts show reactions in control cattle inoculated with the cattle virus only.



KANSAS STATE COLLEGE OF AGRICULTURE AND APPLIED SCIENCE LIBRARY JUN 4 19 MANHATTAN

is unnecessary. It was further shown that cattle virus obtained from Muktesar was adapted readily to local goats by passage, with resulting attenuation in virulence to the desired degree of safety.* It is proposed to carry out experiments later to ascertain whether imported cattle of European origin and cross-breeds can also be economically immunised with safety along these lines.

The saving in money thus achieved in the prosecution of active immunisation on a large scale will be a very great one if it is found that the method can be applied successfully in all parts of the province. The only expenditure incurred, outside that on staff, will be that on goats. These animals have hitherto been obtained by us on hire at a cost of about 1*s.* 6*d.* each ; sometimes they have been purchased, and then sold after recovery (which is usual in field conditions). The financial impracticability of applying active immunisation on a large scale in the province by means of the ordinary serum-simultaneous method of inoculation has been explained by reason of the prohibitive cost of the quantity of serum prescribed.†

The work of immunisation by this method is now in active progress, and it is hoped to submit a further report for publication in six months. Meanwhile, as a precautionary measure, the officers in charge of these experiments have always been provided with supplies of anti-rinderpest serum, with instructions to inject it intravenously in appropriate doses, should the reactions to either goat virus or cattle virus prove alarming. No necessity for the use of serum in this way has so far arisen.

If any field worker is already doing, or proposes to carry out, similar work in India, I shall be grateful to him if he will either publish his results early or communicate a summary of them to me, so that the progress of the work may not be impeded through accidents resulting from lack of knowledge, of which sufficient may become available early to isolated workers.

*Acknowledgments.*—Besides the two colleagues whose names have already been mentioned in this paper, special mention has to be made of the services rendered by Mr. G. D. Pani, who has carried out most skilfully and carefully most of the experiments recorded, and by Rai Saheb R. V. Pillai, deputy director of Veterinary Services, Central Provinces, who has collected for publication the information contained in the field reports received from numerous and varied sources. In conclusion, my thanks are due to my friend and former colleague, Dr. J. T. Edwards, who first inspired me with the idea of conducting field work along these lines.

* Later experience has shown that cattle virus can be fairly easily adapted to goats by subcutaneous inoculation, and thereafter passaged from goat to goat ; the number of failures has amounted to less than 50 per cent.—R.F.S., 18.4.32.

† It has since been estimated that the cost of active immunisation of cattle by the goat virus alone method amounts to about 4d. per animal inoculated, as against approximately 2s. 6d. by the serum-simultaneous method.—R.F.S.

REFERENCES.

(1) Edwards, J. T. "Rinderpest: Some Points in Immunity." *Trans. 7th Congr. Far Eastern Assoc. Trop. Med.* (Thacker's Press, Calcutta). 1927. 3, 707.

(2) Edwards, J. T. "The Problem of Rinderpest in India." *Imp. Inst. Agri. Res., Pusa (India), Bull. No.* 199 (*Govt. of India*), Central Publ. Branch (Calcutta). 1930.

# SOME OBSERVATIONS ON THREE COMMUNICATIONS DEALING WITH THE TREATMENT OF GASTRO-INTESTINAL STRONGYLOSIS OF SHEEP.

By P. L. LE ROUX, B.Sc.(Edin.), M.R.C.V.S.,
*Assistant Veterinary Research Officer, Mazabuka, N. Rhodesia.*

THE treatment of sheep for gastro-intestinal strongylosis still receives considerable attention in various parts of the world, and is of the utmost importance to the sheep industry of Australia, South Africa, and other sub-tropical countries. Publications on this subject by Seddon and Ross (1929), Parkin (1929), and Wood (1930), have been contributed from Australia, South Africa, and England, respectively. A critical study of these communications indicates certain discrepancies which cannot be readily overlooked.

When the vermicidal action of a drug or a mixture of drugs is tested against any parasite, it is only reasonable to expect that the sheep selected for the test should be definitely known to be infested, and that the degree of such infestation should be gauged with fair accuracy. Seddon and Ross and Wood did not take these precautions, and this detracts from the value of their reports. They selected sheep which they believed to be infested. The Australian contributors did not infect their animals experimentally, "since it was not found practicable to infect sheep artificially." Artificial infestation of sheep should always be resorted to when the vermicidal property of any drug or proprietary remedy is to be tested against any particular species. The degree of infestation before and after the treatment of the animal can be readily ascertained by the "cultural" method. The various genera, *e.g.*, *Hæmonchus*, *Œsophagostomum*, *Monodontus*, *Gaigeria*, and *Trichostrongylus*, are differentiated by the unequal lengths of the tails of their larval stages.

The slaughter of treated animals for ascertaining the resultant action of the drug is not an economical proposition in all countries, and is not necessary. The "egg count" method of ascertaining the result is most laborious and unreliable, because the eggs of most *Trichostrongylidæ* inhabiting the gastro-intestinal tract of sheep are very much alike.

The reports from Australia and England refer *inter alia* to unsatisfactory results obtained with the use of the mixture of copper sulphate and sodium arsenite, the mixture recommended by Veglia (1918), against *Hæmonchus contortus*. The authors refer to the findings of Veglia, but their conclusions would seem to suggest that Veglia's results must be accepted with caution. They do not openly state that they doubt his findings. They are not entitled to compare their findings with those of South African workers, since they did not administer the mixture as recommended by him. Their failures to obtain the results claimed by him should probably be attributed to the fact that they administered the mixture in capsules to sheep. This means that the chemicals entered the rumen,

XUM

but not the abomasum, and the results were similar to those obtained when Veglia administered it intra-rumenally by an injection through the left flank. The Australians mentioned the fact that Veglia records two cases where a considerable number of wireworms were not expelled by the prescribed treatment. These were in animals one of whom had an impaction of the rumen, and the other an impaction of the abomasum. My personal experience in dosing sheep with tabloids or with drugs enclosed in capsules is that tabloids and capsules invariably pass into the rumen. Bulky powders follow a similar course. It is not evident why Wood (1930) should recommend that the powdered copper sulphate be given in capsules. This is undoubtedly a tedious procedure where hundreds of animals are to be dosed every three weeks.

The procedure recommended by Veglia enables four attendants to dose approximately 400 or more sheep per hour. Furthermore, the giving of drugs in capsules would amount to mixing them with the food, as they will pass to the rumen, and it is therefore significant to note that Wood tried drugs in food with disappointing results. Where Seddon and Ross used copper sulphate in solution, they claim that some wireworms were killed. The Australians claim the better results when a preliminary starvation preceded the administration of the mustard and copper sulphate in solution draught.

From data personally gathered during the last five years, I can substantiate Veglia's findings as to the efficiency of the Union Government wireworm remedy when administered as originally recommended in the instructions issued to farmers. Recently Mönnig (1929) recommended a very much reduced starvation period, and there were complaints from farmers who had adopted this that the treatment was not as efficacious as before. This reduction of the starvation period was recommended because it was believed that the starved animals would partake more readily of poisonous herbage when returned to the pastures. It was thought desirable to ascertain the efficiency of the remedy in starved and non-starved animals, experimentally infested with *Hæmonchus contortus*. The results which are available show that a preliminary starvation of from 18–24 hours is essential when the maximum effect is to be obtained. The double dosing originally prescribed by Veglia was completely effective in removing all worms. Double dosing proved rather drastic in heavily infested animals. It is desirable that the mixture used in South Africa be given a trial in countries where the wireworm is claiming many victims. Seddon and Ross have rightly remarked that there are no reports except that of Sheather (1927) dealing with the value of the Union Government remedy against *Hæmonchus contortus*. Further reports will be eagerly awaited, but will only be of value for purposes of comparison if conducted as outlined by Veglia. Finally, it may be stated that the efficiency of the Union Government wireworm remedy (termed " Theiler's remedy " by many farmers) will depend on whether it reaches a comparatively empty abomasum directly on administration. This remedy is undoubtedly the best vermicide known for minimising the annual ravages of *Hæmonchus contortus*.

XUM

As regards the other anthelminthics tested by the authors concerned, I can only add that Carbon tetrachloride proved to be no better than the remedy recommended by Veglia. My experience is that 100 per cent. effective drugs are still to be discovered for the numerous species of *Trichostrongylus*, *Ostertagia*, and *Cooperia* parasitising the sheep in various parts of Southern Africa. Carbon tetrachloride was found most efficacious in outbreaks of hookworms (*Monodontus trigonocephalus* and *Gaigeria pachyscelis*).

The publication by Parkin (1929) deals mainly with the possibility of the intra-abomasal administration of drugs in sheep. The author describes his method, and seems convinced that it is a practical proposition. It may be so in the hands of the experienced operator, but not as a general procedure. He undertook his experiments in an attempt to remove *Œsophagostomum columbianum*. The absence of *O. columbianum* at the post-mortem of sheep No. 32 I would prefer to ascribe to the enteritis present rather than to the direct action of the Union Government wireworm remedy administered intra-abomasally. In the case of sheep No. 39, experiment No. 26, he claims that 5 c.c. tetrachlor-ethylene had a positive effect on the nodular worms. This cannot be accepted, for he admits that a "cultural" examination was not made. This animal was therefore not definitely known to be infested at the time of treatment. He does not report whether the drugs introduced had any effect on any of the other parasites present in the gastro-intestinal tract at the time of the experiments. This author also states that the treatment of œsophagostomiasis has up to the present not been crowned with success. This is not absolutely correct, for Veglia (1928) reports very favourably on the enema treatment on experimentally infested sheep. The results were so satisfactory that this is to-day the method recommended to farmers for combating this malady in the Union of South Africa. It has proved very efficacious against nodular worms of sheep at this institute.

REFERENCES.

Parkin, B. S. (1929): "Intra-Abomasal Administration of Drugs to Sheep," *15th Ann. Rept. Dir. Vet. Services*, Vol. 2, Sect. 9, October.

Seddon, H. R., and Ross, I. Clunies (1929): "Observations on the Treatment of Parasitic Gastro-Enteritis in Sheep," *Vet. Res. Rept.*, Dept. Agric, N. S. Wales, Sydney.

Wood, W. A. (1930): "Treatment Experiments on the Treatment Parasitic Gastritis in Sheep and Lambs," *Jl. Agric. Sc.*, Vol. 20, Pt. 2, April.

Veglia, F. (1918): "Chemotherapy of Hæmonchosis in Sheep. *5th and 6th Rept. Dir. Vet. Res.*, April, 1918.

Veglia, F. (1928): "Œsophagostomiasis in Sheep" (Preliminary Note). *13th and 14th Rept. Dir. Vet. Educ. and Res.*, Part 2, October.

---

# FLUKE IN SHEEP.

By WM. A. CAMPBELL, M.R.C.V.S.

*Boroughbridge.*

SEVERAL of the Yorkshire papers have alluded to the serious outbreaks and heavy mortality amongst sheep due to fluke in this district. They talk of it being the worst for thirty years. I think it would be correct to say it is the worst in

living memory. The oldest farmers in the district have never seen nor heard of much death from fluke. I have practised in the district for thirty years, and up to this season always considered it very free from fluke. One client, whose flock has been almost wiped out, never saw the complaint before, and he and his father have farmed the same land for sixty years or more.

The symptoms and general course the disease has run has been altogether different from what I have seen previously, and as for the life-cycle of the fluke worm, I am afraid in my cases there must have been a short circuit. During November and December I opened many carcases dead from fluke that were full of fat and in good condition. The usual history was that the sheep were noticed to hang back from the trough, and in twelve hours they were dead. Scores were slaughtered on showing the first symptoms, and passed for human food. In examining a flock just beginning to go wrong, one had to be careful, as they all looked well and showed none of the common symptoms, but if you drove them for a few hundred yards you could soon see all the sick sheep lag behind, drop their ears, drink at every pool of water; and lots would lie down and refuse to rise. Dropsy of the abdomen is an early symptom, and a fatal one. Many of the sheep that reached the emaciation stage, even with the baggy jaw and blabby eye, recovered with treatment, but hundreds of sheep appeared to be so heavily infected that they died suddenly before any medicinal treatment could be effective. Then, again, many died after treatment, and on post-mortem few or no flukes could be found; but the livers were so extensively diseased, enlarged and cirrhotic, that they could not live.

As regards the life-cycle of the fluke worm, I have had several instances where healthy sheep went on to infected land and started dying within the month from fluke. In one instance twenty healthy shearling gimmers were sold in a local auction about October 21st—when I say healthy, the seller bred them and kept forty of the same lot together with some older ewes—and he has not lost a single sheep up to date. They were bought by a farmer who knew the sheep, and taken to his farm some eight miles away, and mixed with some other ewes, and two rams put to them. Before the end of November they started to die, and every one died. By the end of December that flock of 110 ewes was wiped out; many of the carcases were salved at a bad price to the butcher. His two rams died early, both big, fat sheep. He borrowed a ram off a friend to finish the work, and kept him three weeks. Four days after returning him he died from fluke, and he had been twice dosed. That was the only sheep ever known to die on that farm from fluke.

This owner, who never saw fluke before, argues that those twenty gimmers fetched the disease on to his place. Of course, I do not agree; certainly one or two of them were the first to die, but a few days after, his own sheep bred on the farm started to die, and went quite as rapidly as the bought-in gimmers. It surely takes some explaining how clean healthy sheep can go on to land in November and December, and die wholesale from fluke within a few weeks.

XUM

As regards treatment, I have used male fern as supplied by Parke, Davis & Co., and with good results on the whole. When one saw the livers in scores of cases, treatment of any sort looked useless. I have not used carbon tetrachloride very much, because almost every farmer is buying it at the local chemists and on open market stalls. I cannot say I have seen any great results from its use. I do not know, but the capsules I see appear to me to be too small to hold a full dose of carbon tetrachloride. In many cases the laboratory has told me that sheep dead from fluke are heavily infested with strongyles, so that drenching with cupri sulp. alternately has given good results.

## LIVER FLUKE.

By R. HUDSON. F.R.C.V.S.
*Retford.*

FORTY-TWO in-lamb ewes were grazing on land seeded down about six years ago. The same ewes grazed the same fields the previous summer. It was land unfit for sheep in winter, but sufficiently well drained to be safe in summer. There had not been any case of fluke on this land before. About the first week in September, 1931, the heavy rain caused flooding of the area, and washings from drains and ponds over the fields in question. During October the first loss occurred, and the ewes were dosed with carbon tetrachloride capsules. They were dosed again a month later, and several weeks after that several ewes showing symptoms of fluke were dosed again. These ewes, as the owner expressed it, "went yellow, and died straight off." Deaths continued up to February, 1932, and now total forty out of the forty-two. This owner does not think much of carbon tetrachloride.

I only made one post-mortem, *viz.*, in February, when I found mature flukes in the liver. The owner, a butcher-farmer, could not find them in early cases unless sections of liver were squeezed into water, but has found them in all the ewes he has examined. The remaining two have since died.

## A FEW NOTES ON SOME OF THE COMMON SHEEP DISEASES MET WITH IN YORKSHIRE.

By LIEUT.-COL. H. G. BOWES, F.R.C.V.S.
*Lecturer in Veterinary Hygiene, Leeds University.*

IN selecting the above title, I do not suggest that sheep diseases in Yorkshire differ in any way from the diseases affecting sheep in other counties. I am merely confining myself to diseases which I know to be particularly prevalent in Yorkshire, and which are the cause of the most serious losses amongst sheep; diseases with which I have been brought into contact over a period of a good many years. In dealing with these diseases, I shall approach them purely from the clinical point of view. I would say at the outset that probably more than half the losses amongst sheep, particularly on moorland farms, are due directly or



XUM

indirectly to parasitic causes. Of these, the most widespread is strongyle infection of the digestive system. Fluke disease also is the cause of heavy losses, especially in very wet seasons. The losses from these parasitic diseases are, I think it will be admitted, to a greater or less extent preventable, and it is therefore important that consideration should be given to conditions which favour or hinder the development of these parasites. There are, in addition to these parasitic diseases, others which are particularly prevalent in certain parts of Yorkshire, and which are the cause of very considerable losses. Amongst these are certain diseases incidental to lambing, such as the disease known locally as " tremblings," which occurs in lambing ewes and in young lambs, lamb dysentery, navel-ill or crook, swingle-back, etc.

In the case of some of these diseases the losses are certainly heavier than they would be if veterinary advice was sought more frequently in regard to sheep diseases. Unfortunately the value of the individual sheep is so small, especially in the case of moorland sheep, that it is not considered worth while calling in a veterinary surgeon unless the losses are of a more or less epidemic character. Clinical experience of sheep diseases generally is therefore more limited than that of the diseases of other farm animals. It is not surprising, therefore, that old-fashioned ideas and superstitions dating back for many generations should still persist amongst farmers as to the nature, cause and treatment of many of these sheep ailments. I need only instance the so-called " double scaup " (double scalp) in lambs in the Craven district, which was believed by farmers to be the cause of a wasting disease which they call " drying." This so-called " double-scaup " is simply a thinning of the outer plate of the frontal bone, due to the anæmic condition of the lamb, the cause of which is strongyle infection of the abomasum and intestine, or what is generally known as parasitic gastritis or scour. Incidentally, I may say that a superstitious belief still exists amongst many farmers in the Craven district that affected lambs will not do any good until this "double scaup" is cracked, and it is still a common practice amongst them to crack the "scaup" either with the knuckle, or with a stone. I will now discuss these diseases, and their occurrence in Yorkshire, individually and in rather more detail.

*Parasitic Gastritis.*—This is, I believe, the most widespread disease of sheep not only in Yorkshire but in Great Britain generally. The losses from this disease are chiefly amongst lambs over three months old, but it is also the cause of losses amongst older sheep. It is difficult to estimate the total losses from this parasitic infection, as they are represented not only by deaths, but also by loss of condition and consequent delay in marketing those lambs which are affected but recover. It is quite possible that by lowering the resistance of infected animals, this condition may also act as a contributory cause to other diseases. My attention was first seriously drawn to this disease in sheep some twenty-six years ago, when I was asked to investigate an obscure condition prevalent amongst lambs in the summer months in the Craven district, and known locally as " drying." This, as I have already explained, was at that time asso-

XUM

ciated with, and believed to be due to, the so-called condition of " double scaup." That investigation established the fact that " drying " was simply parasitic gastritis, and that the so-called " double scaup " was due to the wasted condition of the lambs consequent on the parasitic infection.

Parasitic gastritis is so well known that it is not necessary for me to enter into an elaborate description of the disease or of the parasites involved. Suffice it to say that the condition is usually associated with wasting and some diarrhœa, and it may be taken that where lambs over three months old are doing badly, and are scoured, it is most probable that they are suffering from this parasitic invasion. The parasites concerned, of which there are several, belong to the family of strongyles, and without going into any detail as to the different varieties, I would say that in my experience the infection is usually a mixed one of several varieties of strongyle. The one which is usually described as being the more common, *viz.*, *Strongylus contortus*, is the most easily distinguished, as it is much longer and thicker than the others, such as the *Str. ostertagi*, *Str. filicollis*, etc., which are very minute and thin, and are not easily seen by a naked-eye examination of the contents of the abomasum. Confirmatory diagnosis of this disease may be made by microscopic examination of the contents of the abomasum for the presence of the parasites in the dead animal, or of the droppings for eggs and larvæ in the live animal. I hope I may be excused for using the generic term of Strongyles for all these worms, on the ground that I am dealing with them purely from the clinical point of view, and therefore it is more convenient to group all the worms responsible for this particular condition under the one family name. This nomenclature is perhaps somewhat out of date, but unfortunately the more modern names given to these various worms do not indicate the close connection there is between them clinically.

The chief point of interest in regard to this disease as it occurs in Yorkshire is the great benefit which has been derived during the past ten or twelve years from dosing with copper sulphate solution. This is usually given in a 1 per cent. solution in doses varying from one ounce for lambs from three to six months old, to three ounces for adult sheep. On infected farms regular dosing at monthly intervals in the summer, usually in July, August and September, is a common practice, and has materially reduced the losses from this disease. Although recent research into the toxicity of copper sulphate to these Strongyles indicates that it is only toxic to certain varieties, and that it is non-toxic to others, notably *Strongyle ostertagi*, evidence is overwhelming in Yorkshire of the benefit of this treatment in the great majority of outbreaks. The explanation of its success, in spite of the fact that copper sulphate is non-toxic to some varieties of Strongyle, is probably that the infection is usually a mixed one, and that it reduces the number of parasites present to such an extent that the animal is able to resist those which remain. It may even be that the copper sulphate causes the evacuation of some of the parasites without actually killing them. Although this disease is most prevalent in lambs, losses amongst shearlings, and even old sheep, are not

XUM

uncommon on heavily infected land. One case of a moorland farm was brought to my notice last year in which the farmer had lost during the previous winter over 100 ewes chiefly from this disease.

*Liver Fluke.*—I need say very little about fluke disease except that its incidence in Yorkshire during the past winter has been very heavy, as has also the mortality. This has, I believe, been the case in the country generally. There is, however, one fact to be noted, that the losses have been heaviest on farms which had not previously been regarded as being very subject to the disease. On many farms where there is much wet land, and where therefore the disease regularly occurs, it has become the practice to dose all the sheep several times in the late autumn and early winter with carbon tetrachloride capsules, and this appears to have been successful in preventing any serious losses from the disease on such farms. I have not heard of any very heavy losses on farms where this practice was followed last autumn. Many farms, however, which were not regarded as being subject to fluke, either became infected last summer owing to the abnormally wet weather, or sheep were bought in the autumn or early winter which were already infected with fluke. On many of these farms the sheep were not dosed until they began to die, and on post-mortem examination they were found to be badly infected with fluke; this was probably not until about December, by which time the disease was too far advanced, in many cases, for treatment to be effective. I know of several such cases in which more than half the ewe flock died, all of which were on farms where the disease was not anticipated, and where the farmer had not therefore taken the precaution of dosing his sheep in the autumn. There have also been reported to me several instances of deaths occurring after dosing with carbon tetrachloride where the sheep were being fed generously on concentrates, which emphasises the importance of care being exercised in the use of this drug. In such cases it is perhaps safer to use the standardised etherial extract of male fern.

*"Tremblings" or "Moss Illness" in Ewes.*—This is a disease of lambing ewes which is particularly prevalent on moorland farms. It is called "moss illness" by some farmers because they believe that it is caused by the ewes eating a certain plant on the moors which they call "draw moss." This is not really a moss at all, but is cotton grass, and in the late winter the young shoots can be pulled out of their sheaths, and are relished by the ewes, as they are fresh and succulent, at a time when other grazing is scarce. I do not think this grass has anything whatever to do with the causation of the disease, as it sometimes occurs amongst ewes which have not been on the moors at all, and which have not had access to this particular grass. The history of this disease is that it most frequently occurs amongst in-lamb ewes after they are brought down from the moors to the home pastures for lambing. It would appear, therefore, to be influenced to some extent by the change in the conditions of grazing. Sudden changes in the weather, especially cold winds, accompanied by snow or sleet, also appear to have an influence on its incidence.

XUM

The nature and cause of the disease are, however, still obscure ; but it is probable that there are two distinct diseases covered by the term " tremblings," both of which exhibit similar symptoms of nervous movements—nodding of the head, trembling of the fore legs, and finally a more or less comatose condition.

The less fatal form, which more usually occurs after lambing, is probably due to a condition of hypocalcæmia, similar in its nature to milk fever in cows, and this form usually responds readily to either inflation of the udder or to injections of calcium gluconate ; this condition is also known under the name of lambing sickness.

The more fatal form which usually occurs before lambing, and in which the mortality is as high as 80 to 90 per cent., does not appear to be associated with a hypo-calcæmia, and does not respond to injections of calcium gluconate or to inflation of the udder. The term pregnancy toxæmia has been applied to this disease by certain research workers, and although nothing definite is known as to its nature, it has been suggested that there is possibly a condition of hypoglycæmia associated perhaps with acidosis. I have been treating cases of tremblings experimentally during the last few years with injections of calcium gluconate. This treatment has been quite successful in those cases occurring after lambing, as has also been the inflation of the udder.

In the cases occurring before lambing, however, the calcium gluconate injections have met with varying results : in the majority of cases the treatment has been quite ineffective, but on certain farms the results have been more favourable. As recently as last week I visited a farm on which there were ten ewes which had not yet lambed, affected in one day. These were all injected with calcium gluconate, and only two of them died, the remaining eight recovered, and several of them have since lambed live lambs and are going on all right. I assume that although these cases were before lambing, they were not cases of pregnancy toxæmia, but of lambing sickness due to a calcium deficiency. It has been suggested that for pregnancy toxæmia, if it is due to a hypo-glycæmia, injections of a solution of medicinal glucose might be beneficial. I have only had the opportunity of trying this treatment on a very few cases up to date, and am therefore unable at present to express any opinion as to its effect.

*Lamb Dysentery.*—This disease is very common amongst newly-born lambs in the Craven district, where it is known as " ray," and in certain seasons is responsible for very heavy losses. The mortality from this disease is very high, in lambs already affected probably as high as 80 to 90 per cent., and on an infected farm in a bad season I have known the losses to be as high as 40 per cent. of the total of lambs born. Lamb dysentery serum (Wellcome) has proved to be a very effective preventive of this disease in Yorkshire. It is also very valuable in the treatment of lambs already infected, a very large percentage recovering under the serum treatment. In the lambing seasons of 1929 and 1930 I carried out an experiment on a limited number of farms on which half the lambs were injected

XUM

with serum and half kept as controls, the result showed very clearly the value of the serum as a protection against this disease.

*Navel-Ill in Lambs.*—Although this is not so general as some of the diseases already mentioned, it is the cause of quite serious losses on some farms. In the East Riding it is known as " crook," and some farmers lose as many as twenty or thirty lambs a year from it. It may be assumed that it is a form of septicæmia in which the channel of infection is usually the raw navel ; there is swelling of the joints, often accompanied by suppuration. Affected lambs may linger on for weeks, crawling about on the ground, but eventually, if they do not die, they have to be destroyed. A very simple but effective method of preventing this disease is to dress the navel soon after birth with a little powdered copper sulphate. This not only disinfects the navel and cord, but shrivels it up, so that there is no raw surface left through which organisms can gain entrance. Many farmers who have had trouble with this complaint for years have found that this treatment has completely checked it.

*Swingleback or Swayback in Lambs.*—Finally, I would like to refer briefly to this mysterious complaint. On some farms a certain proportion of the lambs when they are born are paralysed in the hind limbs, these lambs seldom do any good, and eventually have to be destroyed. It appears to be much more prevalent some seasons than others. Last year quite a lot of lambs were born affected in this way in certain districts of Yorkshire. It has been suggested that it might possibly be associated with a mineral deficiency, and one farmer in the North Riding who had in previous seasons lost heavily from this disease, acting on the assumption that it might be due to a mineral deficiency, two years ago fed a supplementary mineral mixture to his ewes throughout the winter. The following spring 50 per cent. of his lambs born were " swinglebacked " and had to be killed. It is unlikely from this evidence, therefore, that it is due to a mineral deficiency. It certainly would appear to be a disease which merits further investigation.

The foregoing are a few of the more important troubles which confront the sheep farmer in Yorkshire ; there are many others, of course, but these are, I think, the ones which cause him the greatest concern.

---

## CŒNURUS CEREBRALIS, OR GID, IN SHEEP.

By CHAS. GILLARD, M.C., M.R.C.V.S.
*Folkestone.*

It was while practising at Ashford some years before the war that I became particularly interested in this disease. I then collected a series of very interesting photographs and records of a large number of sheep so affected. Unfortunately most of these have been lost, and I only have by me three or four photographs and occasional notes. My particular reason for choosing this subject is because I believe it has been much neglected by veterinary surgeons, and I should like to

encourage members of the profession to devote their attention to the clinical aspect. They will find it remunerative, and even more interesting than the life history of the causal parasite, and will have the satisfaction of lessening the financial loss of sheep farmers. Practically all text-books discourage any treatment, and recommend early slaughter of affected sheep, whereas there are many hundreds of these cases which make ideal subjects for surgical treatment. "Gid" is also known under the names of sturdy, staggers, turnsick, etc., and in Kent, "pothery." It is a disease which has been known as affecting sheep for at least some 300 years. In 1634, two European surgeons discovered in the brain, on post-mortem examination of a sheep suffering from vertigo, "a vesicle filled with fluid." In 1780, Leske recognised the bladder as a cystic worm, but not until the middle of the last century was its true nature discovered, and tape-worm heads found. In 1852, Seibold proved that the bladder worms when fed to dogs developed into mature tape worms in their intestines. In 1853, Keuckenmeister demonstrated that tape-worm eggs from dogs, when fed to sheep, produced bladder worm in their brain and spinal cord. These observations were afterwards confirmed by many workers, and so it was established that the *Tænia cœnurus* of the dog and the *Cœnurus cerebralis* of the sheep were the laval and adult forms respectively.

Statistics quoted in various text-books agree more or less as to percentage of sheep attacked, *viz.*, in the first year of life from 2 per cent. to 5 per cent., in the second year, 1 per cent. to 1½ per cent., afterwards only occasional cases. But these figures do not take into consideration the occasional outbreaks, when anything from 15 per cent. to 35 per cent. will be affected. These numbers in themselves are sufficient to emphasise the importance of this disease from an economic standpoint, but when one bears in mind that these losses can occur in pedigree flocks, the financial loss to the sheep farmer may be most serious.

Infection takes place, in most cases, in early life, when the lamb is only a few months old (due probably to less power of resistance) owing to ingestion of the proglottides or matured ova of the *Tænia cœnurus* voided by the dog.

*Types of Cases.*—Broadly speaking there are two distinct types, *viz.*, cerebral and cerebellar. In each type there will be some signs common to both, *e.g.*, altered facial expressions, ear carriage, unsteadiness of gait, grinding of teeth, swaying movements, exaggerated action of one or more limbs, varying degrees of paralysis, and loss of flesh. In the cerebral type, as the cyst matures, the senses are dulled, sight, hearing, touch, or all three. The head is held differently, either to one side or slightly raised or depressed, one or both ears may be lopped; the sheep will be generally less inclined to move about, excepting the animal which has the cyst located in the extreme anterior portion of the cerebrum, involving the olfactory lobes, when it constantly walks more or less in a straight line until meeting some obstruction, and there it will remain for a long time (*see* Fig. 2). Many appear quite stupid, and do not attempt to run away when approached by man or dog; a large percentage show complete blindness in one eye, some

XUM

show paralysis of the lips, accompanied by excessive salivation, which may be green in colour (termed " greenmouth " by some shepherds). On closer examination many of these cerebral cases will show softening of the cranial bones, but one must be careful not to presume this to be always associated with the presence of a cyst: it is sometimes due to injury, in which case there is a depressed fracture of the skull. Then there are the characteristic turning or circling movements to left or right when grazing, and when the head is held at the normal height when moving to fresh grass; and as the cyst grows, and more brain tissue is injured, the sheep is unable to stand, and finally there is general paralysis or death from exhaustion. The cerebellar cases, on the other hand, are characterised by quite a different set of symptoms: absence of softening of the cranium, no blindness, no expression of stupidity; but there is marked hyper-æsthesia, and on seeing either a man or a dog approach, such an affected sheep darts off at once, holding the head somewhat higher than normal, and directed to the opposite way in which the rest of the body is travelling. There is a frightened expression, a stilt-like gait of the hind legs, as in Fig. 3. As these cases become more advanced, the staggering gait becomes more marked, and in attempting to get away the sheep does so with jerky movements: it falters, falls over, struggles violently, regains its feet, repeats the performance, ultimately collapses entirely.

*Treatment.*—The only treatment is surgical, *viz.*, boring or trephining the skull, removing the cœnurus bladder or cyst and fluid contents (refer to inflammatory or meningeal fluid); but before this can be carried out the location of the cyst must be decided upon. At least 75 per cent. of the cerebral type can be successfully operated upon, and if one selects the more favourable cases, *viz.*, those which show very decided circling movements with accompanying blindness in the eye opposite to the side which the sheep circles, also cases showing marked stupidity and dullness, with softening of the skull over the olfactory lobes or just behind the horn core, then 85 per cent. to 95 per cent. of cures can be expected. Though I have successfully removed the vesicle from the posterior brain, I do not advocate attempting operative treatment in the cerebellar type, owing to it being nearly impossible to avoid fatal hæmorrhage.

*Selecting Cases for Operation.*—Whenever possible arrange to have the affected sheep penned up and fasted over night, then when turned on to grass the sheep will at once commence to graze, provided the case is not too advanced, and in the space of a few minutes one will notice the characteristic symptoms already mentioned. The commonest variety are those which when grazing do so in either small or large circles, and when seeking fresh herbage, with head held in normal position, walking round to same side forming half, one, or more complete circles. Others of this type will hold their heads directed round to the shoulder of the same side; practically all these cases will show blindness in the opposite eye, and the best way to test this is to quietly walk up to the sheep as it is grazing, or standing still on the opposite side to which it turns, holding a white handkerchief or other object close to the eye, when it will at once be observed the sheep takes

XUM

FIG. 1 *(see page 216).*

Shows cyst on the floor of the 4th ventricle, and collapsed position after attempting to walk.

FIG. 2 *(see page 215).*

Shows cyst in olfactory fossa causing the sheep to keep on walking more or less in a straight line. In the photograph the affected sheep is unable to avoid butting into another.

FIG. 3 *(see page 216).*

Cyst occupies right posterior part of cerebellum. The head is carried slightly to the right and the hind parts to the left in attempting to walk forward.

XUM

XUM

no notice, while on passing the hand over to the other eye the sheep will instantly jump away from one's hand. The skull is then examined for sensitiveness to pressure, and for a softened area, which there will be in the great majority of this type if not more than fifteen months old. In a good many of these the skull will have been very much thinned by the pressure of the cyst, or by the action of the fluid when the vesicle has burst. An important feature to notice in this type of case is that when such a sheep is driven into a corner, and when attempting to escape, it will invariably swing sharply round to the opposite side to which it circles, and a certain number will show keratitis, the result of injuries to the blind eye. These symptoms of circling, blindness, etc., denote the position of the cyst, which will be found occupying the area of the lateral ventricle on the side to which the sheep circles, or in other words, the opposite side to the eye which is blind. I might here remark that in the cerebellar type the affected sheep invariably walk away from the side where the cyst is located, and also falls on to the opposite side. Fig. 2 illustrates the type which keeps on walking already referred to. In this case it has blundered into another sheep, and as the latter did not move away, the sheep in question remained in that position for a considerable time.

*Operative Technique.*—Having made up one's mind as to the position of the cœnurus cyst, the sheep is placed upon a table, all legs tied together in the fully flexed position by means of a piece of stout cord, held by an assistant, a second holding the sheep's head; the wool is clipped off the skull, and the immediate seat of operation closely clipped with scissors; the skin, if very dirty, is washed with æther soap and warm water, otherwise treated solely with tincture of iodine. One c.c. of codrenine or other suitable local anæsthetic is them injected into the skin immediately over the spot where one imagines the cyst to be: generally speaking, half an inch to one inch behind the horn core. Strict asepsis must be observed, a small saucepan or steriliser should be close by, and any instrument used should be put back into the boiling water before re-using. A short incision is made through the skin, artery forceps applied to any severed vessels for a moment, to prevent blood entering the cranial cavity, and the skull is penetrated by means of a trochar or trephining borer, or by a small trephine. Personally, I rely upon the former when there is softening of the skull, and the borer when this condition is absent. When the borer is used it is advisable to remove the powdered bone before entering the cranial cavity. The *dura mater* must be punctured, and if the cyst is on the surface of the brain some portion of it will instantly protrude, especially if the sheep struggles or jerks its head; and in a good many cases a considerable portion of the vesicle will push its way out, but it sooner or later bursts. If the cyst is not yet visible, carefully introduce a small cannula for about a quarter of an inch, by means of a small syringe endeavour to extract the cyst by suction. Brass syringes are advertised for this purpose, but it is much safer to use a glass syringe, as one can then readily notice if any fluid is being withdrawn, and if this is quite clear, or tinged with blood; if

XUM

the latter, discontinue use of syringe and introduce a grooved director, then carefully probe for the cyst, and if this has already collapsed, ascertain extent of cavity, re-insert the cannula, this time a little deeper, again carefully use the syringe, and with patience one will invariably bring some portion of the cyst wall into the bore hole. One should then secure some portion of the membrane, and by exerting traction at first at one place and then at another, the cyst will be detached and extracted. Now attempt to remove the fluid by again using the syringe, but with great care, otherwise hæmorrhage will take place; empty the fluid as the syringe is each time withdrawn into a measure glass, as the amount withdrawn should coincide more or less with the size of the cyst removed. Small cysts will yield half to one ounce, whereas large ones yield two and a-half ounces. An ordinary milk syphon in many cases will be found useful, as one can, while the syphon is in position, manipulate the sheep's head by turning the nose to either side, or downwards or upwards as the case may be, and so draw off the fluid. Nearly all writers on this subject advise not removing all the fluid at the time of operation, but in my opinion all the fluid should be removed. All that now remains to be done is to apply a little iodine to the wound, cover up the bore hole by pushing back the periosteum, introduce a single suture into the skin wound, apply more iodine, dry the surrounding parts if bloodstained, especially in hot weather to avoid the attentions of the fly, release the sheep's legs, and allow it to go free in the field or paddock, but avoid turning out where the sheep might get into a ditch or pond. In advanced cases where the cyst has pressed upon the olfactory region there is a great tendency for the sheep to walk down to the edge of a pond or wet ditch, and many have been drowned in a few inches of water through merely pushing their noses under the water. Never operate at or close to the middle line of the skull, otherwise fatal hæmorrhage will ensue if the longitudinal fissure is penetrated.

*Recovery.*—When once the cyst and fluid have been removed, the return to normal is most marked in a short time: even within half an hour sheep which have not stood for several days are again able to do so, and in about two hours will be walking. Sheep recover their sight sometimes within twelve hours. I should have said something about differential diagnosis. Gid may be mistaken for injuries to the brain, chiefly depressed fracture, especially in quite young tegs. Again, the greenmouth sheep already referred to is frequently the victim of *Œstrus ovis* larvæ, which also cause extreme dullness. There is, however, generally some nasal discharge.

---

## WOOL-BALL IN LAMBS; MILK CLOT; SAND IN THE ABOMASUM.

By R. HUDSON, F.R.C.V.S.
*Retford.*

One reads very little about the above conditions in text-books, but, nevertheless, they are a cause of considerable loss to flock owners. I would regard it as

being most common among the heavier breeds, probably because they have reached a higher state of domestication, yielding more and richer milk, and having less opportunity and inclination to take the exercise which the lighter hill sheep gets. As lambing time approaches the ewe flock is brought nearer home, and receives a more liberal ration, with the result that the milk supply is forced and enriched beyond the lamb's capacity to digest it. Casein is precipitated, and only partly dealt with by the gastric juices. Some remains in the abomasum, and is added to, forming itself into an insoluble mass which cannot pass the pyloric opening. One can imagine this solid curd giving rise to a feeling of indigestion which causes the lamb to pick up wool and lick sand, perhaps in search of alkali to relieve the condition, but usually making it worse, the wool binding it, and the sand irritating the mucous membrane, or accumulating and often blocking the pylorus.

The symptoms are usually pretty well defined, and easily recognised by the shepherd. The lamb is dull and listless ; when roused it will make for the teat and suck, is soon satisfied, often staggers away and lies down ; later it moves stiffly. The abdomen becomes tight, respiration increases, ears become cold, peritonitis and pneumonia set in, to be soon followed by death. Sometimes the lamb is found dead without any symptoms having been observed, and others may show signs of fits, caused by irritation of the stomach and plugging of the pyloric opening.

Treatment should be more concerned with flock management, for it is doubtful if anything can give relief to a lamb showing symptoms except removal of the insoluble contents of the stomach. Should such aid be sought, however, the opening of the abomasum offers a fair chance of success. Two out of three lambs recovered, and the one which died lived a week before it succumbed to pneumonia.

*Operation.*—The skin is mostly dirty and needs thorough cleansing, wool removing, and dressing with iodine. About 2 c.c. of 1½ per cent. sol. cocaine is used as an anæsthetic. An incision about two inches long is made a little to the left of the middle line and one or two inches behind the sternum. The abomasum is often distended and pressed into the wound, so care should be taken to avoid puncturing it. It should be carefully drawn out, laid on a cloth, sterilised in antiseptic, and incised with as small an incision as necessary for the evacuation of its contents. Sand is the most difficult to remove, but as it is usually in mass at the pylorus, it can be everted through the hole.

Suturing of the abomasum is more quickly performed by a continuous suture, using fine fishing gut—Jagut No. 0—and a number of interrupted Lembert sutures as support. Muscles and peritoneum are taken together, using interrupted sutures, and taking care to close up the opening well because the skin wound around is very prone to suppurate owing to infection and the difficulty in keeping any dressing in position. Tinct. iodine is applied to the wound. After-treatment depends on the lamb. If active, it can return to the flock, if not, and it needs bottling, strong hay tea is better than milk for a day or two, and later milk and water.

XUM

# THE POSSIBLE ASSOCIATION OF THE SHEEP NOSTRIL FLY (ŒSTRUS OVIS) WITH PNEUMONIA IN SHEEP.

By NORMAN BISSETT, M.R.C.V.S.

*Veterinary Advisory Officer for South Wales and Monmouthshire, University College, Cardiff.*

THE sheep nostril fly (*Œstrus ovis*) is not usually considered, either academically or agriculturally, as contributing in any noteworthy degree to the rate of ovine mortality. The literature on the parasites dismisses the effects of the larval infestation with the statement that deaths are uncommon. When they are recorded they are described as being accompanied by symptoms of meningitis (false "gid"). While a certain proportion of farmers are acquainted with the "grub in the head," they do not seem to be aware of what it actually is, or how it came to arrive in that situation. Some observations of the writer would appear to show that *Œstrus ovis* is responsible for a fairly high percentage of losses among sheep, if not annually, at least comparatively frequently. Before these observations are detailed, it is advisable that a brief summary of the life-history of the fly be given.

*Summary of the Life-History of Œstrus ovis.*—The adult fly, which superficially resembles a honey bee, is on the wing during the months of July, August and September. During fine, bright weather, eggs and actual larvæ are deposited in the nostrils of sheep, which may be seen attempting to evade attack by keeping their muzzles close to the ground, or by rushing about in a frightened manner. The larvæ immediately begin to wriggle their way up the nasal passages to the sinuses in the skull just in front of the brain. There they attach themselves to the lining membrane, and remain until they are full grown—a period of between eight and ten months. Their length then measures 25 to 30 millimetres, or approximately one inch. Between the months of April and July the larvæ leave the sinuses and wriggle down the nasal passages, falling, or being sneezed out on to the ground, where they bury themselves and pupate. In this state they remain for from three to six weeks, depending on weather conditions. Finally the fly emerges from the chrysalis, and the cycle recommences.

In the light of the above description, the following observations are interesting: In May, 1930, the writer investigated an outbreak of pneumonia among sheep on a farm in Glamorgan. Fifteen animals had died before the attention of the Department was drawn to the problem. A post-mortem examination was made of one particular animal, which previous to slaughter showed a profuse muco-purulent nasal discharge, and all the clinical symptoms of pneumonia. The examination revealed a condition of broncho-pneumonia. The trachea contained a large quantity of blood-stained tenacious mucus. In this mucus an œstrus larva, measuring 25 mm. in length, was found at the bifurcation of the bronchi. Despite its size and brown colour, it was difficult to distinguish in the discoloured

XUM

material in which it was enveloped, and it was as the result of its movements that it was first noticed. Further investigation resulted in the discovery of another larva, 15 mm. in length, which had made its way into a subsidiary bronchus as far as its dimensions would permit, where it lay in a welter of muco-purulence. No more larvæ were found in the bronchi or their divisions, but eight were removed from the frontal sinuses. The remainder of the flock was then rounded up and examined. A number of animals showed a discharge from the nostrils, and other symptoms of incipient respiratory trouble, but none was so serious as the case which was slaughtered for post-mortem examination. Instructions were given for attempts at expulsion and eradication of the parasites, but in spite of all efforts, three more animals died before the end of June. In these cases the pneumonia was definitely septic, and was of the lobar type, involving practically the complete lungs. In none of the three instances were larvæ found in the bronchi or lungs, but they were present in the frontal sinuses in the proportions of six, twelve, and fourteen respectively.

Between May and the end of June, six outbreaks of pneumonia similar to the above were investigated on as many widely separated farms in Glamorgan and Monmouthshire. In all twelve post-mortem examinations were made. In seven instances larvæ were found in the bronchioles, while in the remaining five cases none could be discovered in these sites. It is especially interesting to note that in the latter group the type of pneumonia was lobar and septic (the chief invading organism being apparently *B. pyocyaneus*), whereas in the case o the group in which larvæ were found in the bronchioles, the pneumonia was of the lobular or bronchial type, *i.e.*, not so far advanced.

The measurements of the larvæ thus found, excluding those first mentioned, varied from 4 mm. to 10 mm. None approached the dimensions of those first encountered ; indeed, had it not been for the experience of the first case, it is quite possible that they might have been overlooked owing to their pale colour, their small size, their penetration into the smaller bronchioles, and their envelopment in the discoloured tenacious mucus. In every case but one, two larvæ were found ; in the exception, only one was detected. The smallest number found in the frontal sinuses was six, the greatest twenty-three. They were present in these sites in every instance, whether or not they were discovered in the lungs. In none of the affected flocks were any symptoms of brain trouble noticed in spite of close observation.

The above discoveries were of particular interest to the writer. In the first place, so far as he has been able to find in the literature on the subject, the presence of the larvæ of *Œstrus ovis* in the lungs (*i.e.*, the bronchioles) is a new record. In the second and more important place he has periodically encountered outbreaks of ovine pneumonia at this season (May and June) for some years past, without having been able to assign a definite cause. A characteristic feature of the outbreaks was the tendency of the condition to become septic, following bacterial invasion.

XUM

Past records were, therefore, consulted, when it was found that while a certain number of cases had occurred every summer since 1923, the years 1926, 1927 and 1930 were notable for the exceptional mortality from this cause. Further reference to records of the Meteorological Office showed that the number of sunshine hours in the area in question, *viz.*, Glamorgan and Monmouthshire, were above normal for the months of July and August—*the month when the adult fly is on the wing*—in the years 1925, 1926 and 1929. The preference of the fly for bright, sunshiny weather for the deposition of eggs and larvæ will be remembered. In view of the foregoing observations, it is very tempting to allow the wish to become father to the thought, and return a verdict of guilty against the œstrus larva without further parley. Let us, however, give the prisoner at the bar a chance to offer what defence he can.

*Arguments for the Defence.*—In exculpation of the larva it may be argued: (1) That no larvæ had been found in the sites in question previous to this year. (2) That those found this year in such sites were very few in number. (3) That in five cases out of twelve no larvæ were found in the lungs. With the most unbiassed mind possible under the circumstances, one is unable to offer further support to the prisoner.

*Arguments for the Prosecution.*—Let us deal in the first place with the above three arguments for the defence, *seriatim*: (1) The fact that no larvæ have previously been recorded in the lungs may be due to lack of observation. As already mentioned, or rather confessed, it was the unexpected discovery of the full-grown larva in the first case that put the writer on the track. Another reason, which may also be applied to defence No. 3, is that the majority of cases were those of the septic and lobar type of pneumonia, and it is quite possible that the larvæ had become involved and destroyed in the inflammatory and septic processes.

(2) The fact that so few larvæ were found does not preclude the possibility of more having been originally present. In any case, the presence of even one active larva living, feeding and excreting in the lung, would be quite sufficient to set up a pneumonic process under ideal conditions for the eventual development of sepsis.

(3) In the five instances in which no larvæ were found, the pneumonia was of the lobar and septic type. The probable reason for non-discovery of the parasites has been given above.

In addition to these arguments, the further significant fact may be adduced, that all cases of this almost epidemic form of pneumonia in sheep recorded from 1923 to 1930 have occurred during the critical months of April, May and June, *when the larvæ are migrating to the exterior*. As above recorded, it has been definitely proved that some larvæ undoubtedly take the wrong turning, and instead of dropping to the ground, pass through the glottis into the trachea, and eventually into the bronchi and their ramifications. It is rather difficult to believe that this was the first year in which they began to forsake the straight and narrow way.

XUM

In this connection, another theory suggests itself. One has been struck by the variation in the size of the larvæ found at the time when theoretically they should all be approaching full growth. The life-cycle commences with the adult fly emerging from its pupal stage in the month of July, whereas, from cases examined in the middle of the month of June, larvæ measuring only 4 mm. have been obtained. Is this the result of pædogenesis (larval reproduction), and an aberration of the migratory instinct on the part of the second larval generation? If so, the position becomes more complicated, as it is of little use attempting to eradicate the parasites by the application of drugs to the upper respiratory passage, if, as the result of pædogenesis, a fresh brood of larvæ is taking up its abode in the bronchi and lungs.

---

## THE VALUE OF OXYGEN ADMINISTERED BY THE SUBCUTANEOUS ROUTE IN OVINE-PNEUMONIA.

By J. S. STEWARD, M.R.C.V.S., D.V.S.M.,
*Hereford.*

THE writer has found the subcutaneous administration of oxygen to be of considerable value in the treatment of pneumonia in animals, and in support of its value in sheep can quote the following more or less controlled experience. During July, 1929, the writer was asked to attend some sheep which were suffering from pneumonia, caused by the inhalation of sheep dip (carbolic). The sheep had been dipped on the Monday, and were first seen on the following Thursday morning, when six of the flock showed great dyspnœa and temperatures of from 103.8–104.5° F. A grave prognosis was given, and treatment consisted of sodium salicylate and camphor in electuary form, and oxygen was administered subcutaneously at the second visit, on the Thursday evening.

A post-mortem examination of two sheep on the second visit confirmed the diagnosis, and the lungs were so solidly hepatised that the owner thought they were the liver; the trachea was full of blood-stained froth, and the tracheal mucous membrane was black from extreme congestion. Oxygen was given to four sheep, one of which appeared moribund; but to one lamb, the least severely affected of the five, it was considered inadvisable to administer the oxygen because of its vigorous resistance, which it was feared might cause death from heart failure; this lamb therefore acted as a control on the oxygen treatment, and it is interesting to note that it was the first of this group to die, whereas the one which appeared moribund at the time of oxygen administration survived it by thirty-six hours; one of the sheep treated with oxygen survived, the remaining three dying in from thirty-six to seventy-two hours after they were first seen. The oxygen was administered directly from the cylinder by the attachment of a hypodermic needle to pressure rubber tubing, and then inserting the needle under the skin and slowly opening the valve; by massage the oxygen was dispersed beneath the skin all over the body, and it was admin-

XUM

istered until the skin was tensely inflated and apparently separated from the underlying tissues by from one and a-half to two inches over the greater part of the body. No evidence of pain or discomfort was noticed: in fact some respiratory relief was quickly evident, and it was from twenty-four to thirty-six hours before the oxygen was more or less completely absorbed, when its administration was repeated as before. From the above and other experiences, the writer considers that oxygen administered subcutaneously is worthy of more extensive use in country practice.

# Reviews

**The Genius of Louis Pasteur.** London: Alexander Ouseley, Ltd. Pp. 361. Price 12*s*. 6*d*.

PERHAPS no one has so greatly influenced modern thought as Louis Pasteur. His researches into the causation of disease and the origin of life led to their universal acceptance, and altered the whole trend of scientific and moral thought. He has been acclaimed the greatest Frenchman of all time, and he is certainly one of the world's outstanding geniuses. The life of such a man has naturally formed a theme for many writers. Perhaps the best biography is that of M. Roux, who, as one of Pasteur's assistants, writes with an inside knowledge and an affection that none can hope to equal. But amongst the others, this new book by Mr. Compton should be awarded a very high place, if only for the genuine affection he displays for the memory of M. Pasteur. One feels that he reveres Pasteur in the same manner as mediæval students of medicine deified the memory of Hippocrates. One also wonders whether Mr. Compton is a Frenchman, for he is at some pains to impress upon us that Pasteur came from that simple French peasantry stock which has provided so many great national heroes, and throughout his book there shines a pride in the lustre which Pasteur has shed upon the name of France. His matter is well arranged. He has grouped it under a series of headings which form a natural sequence. In the opening chapter we are introduced to the parents of Pasteur, and the atmosphere of Pasteur's home is delicately stressed for us so that we shall understand the ideals to which the young man was brought up, and which were responsible for so much of his success in later years. This necessary preliminary step taken, we follow the young man through youth, middle age, to the honours which came to him at last in his old age, and to his death in 1895. Every aspect of his life is dealt with, and we are not even allowed to forget the moral side of his career, the ideals which animated Pasteur, and which he considers were largely responsible for his success. The book can be recommended both to the student who has no time for the more extensive biographies, and to the lay reader who has a taste for national history and is interested in the lives of its foremost workers.

E. A. G.

# Personal

MR. A. NOEL PILLERS, who has contributed on numerous occasions to our columns in THE VETERINARY JOURNAL, has been promoted to be Chief Veterinary Officer to the Liverpool City Corporation in the place of Mr. Eaton Jones, who recently retired. Mr. Pillers has for some years held the appointment of Deputy Chief.

MR. M. R. MAHAGAN, M.R.C.V.S., has been appointed as a Veterinary Investigation Officer to the province of Hyderabad in India.

XUM